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PERIODONTAL DISEASES

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PERIODONTAL DISEASES

Diagnosis and Treatment

BY

ARTHUR H. MERRITT, D.D.S., F.A.C.D., F.A.A.P.

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1930

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TO

DR. GEORGE R. WARNER

OF

DENVER, COLORADO

CLASSMATE AND FRIEND

WHO SINCE THE DAYS WE WERE STUDENTS TOGETHER

HAS BEEN A FAITHFUL CORRESPONDENT, A WISE AND SYMPATHETIC

COUNSELLOR, AND AT ALL TIMES A GUIDE AND INSPIRATION

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PREFACE

THE purpose of this book is to present, in an orderly and comprehensive way, an outline of the principles and practice of periodontia. It is intended primarily for the general practitioner of dentistry and for the undergraduate student, both of whom, it is believed, need help and encouragement in the field of periodontology. For the periodontist it is not believed, and the author dares not hope, that it will possess anything of interest; it teaches no new theories and has no quarrel with old ones. It is an attempt to set forth in plain and simple language the present-day conceptions of periodontia, in the hope that such treatment of the subject may prove helpful to both the student and busy practitioner.

In attempting to do this, the author is not unmindful of the rapid advance that is being made in the field of periodontology. He realizes that some of the observations here set forth as fairly representive of present-day conceptions, may, in the light of tomorrow, be set aside. This is more or less inevitable, inasmuch as there are still unsolved problems in periodontia. Especially is this true regarding those biological factors which, to a certain extent, underlie all periodontal diseases. While these diseases are local in their manifestations, and the dentist in his practice devotes most of his attention to their immediate relief, there can be little doubt that in some cases, at least, they are profoundly influenced, if not actually caused, by what is often referred to as "systemic conditions."

It is not at all improbable, as pointed out by Simonton,

Gottleib, Orban, and others, that there are two types of periodontoclasia: one, a more or less local condition caused by local factors; the other an "atrophy of the bone" due to some peculiar metabolic disturbance. In the present state of knowledge, little of an exact nature is known regarding these biological problems or of their actual relationships to diseases of the periodontium. They are alluded to here, in recognition of their probable importance, and in the belief that their solution will mark an epoch in the practice of periodontology. Meanwhile, and until such time as the research workers in this and allied fields can point out to the clinician the practical application of their investigations, and how through such application he can more successfully meet the demands made upon him by his patients, he must continue to apply himself to the control of the local conditions by methods which, though not perfect, experience has shown are capable of yielding a large measure of success. To set forth these methods clearly and without confusion, so that the dental practitioner anywhere can understand and apply them in his daily practice, is the purpose that has animated the author in the preparation of the following pages.

More and more it is coming to be realized that the maintenance in health of the tissues which support and surround the teeth is the first obligation of the dentist. Most of the diseases to which they are subject are, in large measure, preventable diseases; all yield readily to early treatment, whereas neglect, if long enough continued, may terminate in complete loss of the teeth with all that that involves.

To outline therefore in terms so clear that "he who runs may read," how such diseases may be prevented; what is their etiology and pathology, and how they may be successfully treated, is the self-imposed task of the author. No one realizes better than he that this is a difficult task, and

one which he cannot hope to completely fulfill, but he is encouraged by the reflection that "a man's reach should exceed his grasp, or what's a heaven for"?

If, therefore, his efforts in that direction shall serve to push back ever so little the barriers of ignorance; if they shall help some one to a clearer understanding of the principles and practice of periodontia, and the place which they occupy in the practice of modern dentistry, they will have served their purpose, and the author will have had his reward.

PERIODONTAL DISEASES

CHAPTER I

HISTORY

Diseases affecting the supporting tissues of the teeth are as old as history itself. Frequent mention is made by ancient writers of "loose teeth," "shaking teeth," "hemorrhage of the gums," etc. In the Ebers papyrus, dating from the thirty-seventh century, B.C., certain remedies are recommended "to strengthen the gums." One of these for curing teeth "that gnaw or gives pain unto the gum" is composed of equal parts of incense, verdigris and green lead.[1] There is abundant evidence that diseases involving the periodontal tissues were prevalent among the Greeks, Phœnicians, Etruscans, Hebrews, Chinese and Romans, as they were also among peoples of the Middle Ages. The frequent allusions to "loose teeth" and "bleeding gums," with their discharge of "corruption," all point to the fact that periodontoclasia, as it is known today, was a not uncommon disease in remote times and expressed itself in a pathology not unlike that of the present.

IMMUNITY OF PREHISTORIC RACES

From such information as is obtainable from a study of prehistoric crania, it is probable that primitive man was more or less immune to periodontal diseases in so far as such diseases affected the osseous support of the teeth. This would seem to indicate that in some way not at pres-

ent entirely clear, civilization is responsible for the development of that group of diseases, the tendency of which is to break down and destroy the periodontal tissues. This much at least is certain, that as far back as the written record goes, man has suffered from diseases of the periodontium that have caused teeth to loosen and fall out. Some of the reasons why this should be so will become evident as the story of periodontia unfolds.

Among the writings of the Chinese, some of which date back to more than 2,000 years B.C., frequent mention is made of diseases of the teeth and gums. Among the numerous remedies advocated for the treatment of those cases in which "the teeth often fall out and the breath becomes fetid" is "the urine of a child used as a mouth wash." Another remedy for "pain in the teeth after mastication—excoriation of the gums; flow of purulent mucus mixed with blood; bad smelling breath," etc., was one of especial interest to the present-day student of the subject, in that it contained arsenic. The remedy and its use as described by Dabry consisted of the following: "Arsenic (gr. 1.80) houang-tan (gr. 3.60) pulverize, mix with water, and with a part of the mass form a small pill, which put close to the aching tooth or into the ear, if afraid of the arsenic; then sleep. Cure certain." [1]

Among the Greeks, Hippocrates (about 460 B.C.), the "Father of Medicine," refers to diseases of the periodontal tissues and their treatment in the following words: "When a woman's mouth smells and her gums are black and unhealthy, one burns, separately, the head of a hare and three mice, after having taken out the intestines of two of them (not, however, the liver or the kidneys); one pounds in a stone mortar some marble or whitestone, and passes it through a sieve; one then mixes equal parts of these ingredients, and with this mixture rubs the teeth and

the interior of the mouth; afterward one rubs them again with greasy wool and washes the mouth with water. One soaks the dirty wool in honey and with it rubs the teeth and gums, inside and outside." [1] This is an interesting observation, not only for its reference to diseases of the gums, but in its recommendation of "marble or white-stone," for in this is seen that the use of calcium carbonate or chalk as an ingredient of a dentifrice goes back to antiquity. Many other supposedly modern conceptions regarding the treatment of "loose teeth" will be found to be far from modern, as one ponders "over many a quaint and curious volume of forgotten lore." [4]

The works of Celcus, a Roman, who was born about 30 B.C., have an especial interest in that they sum up in an admirable manner the whole of the medical and surgical science of the ancients from the earliest times down to the days of Augustus. He writes at length concerning diseases of the teeth, including those affecting their supporting tissues. In cases of "loose teeth" he advocates that the gums be touched lightly with a "red-hot iron," after which they are to be smeared with honey and "washed with mulse." When the "gums separate from the teeth" he recommends that unripe pears and apples be chewed and that their juices be kept in the mouth. Equal advantage, it is said, may be had from the use of vinegar, providing it is not too strong. Thus is seen the early use of the cautery and acid mouth washes.

Pliny, another Roman, born in 23 A.D., writes of many potent remedies that are fearfully and wonderfully compounded. It may be interesting to note some of them as they are recorded in "Guerini's History of Dentistry": "The ashes of stags' horn, rubbed over loose and aching teeth, makes them firm and soothes the pain." "The heel of the ox kindled and brought close to loose teeth makes them

firm." "A good dentifrice is also made from the ashes of the feet of a goat." Another dentifrice, according to Pliny, consists of "the filth of the tail of sheep rolled up in little balls, and left to dry and then reduced to powder and rubbed on the teeth; this is marvelously useful against the loosening and other diseases of the teeth." "The ashes of the shells of snails mixed with myrrh, rubbed on the gums, strengthens them." "It makes the teeth firm to pick them with a porcupine's quill." "To make loose teeth firm, some advise the soaking of two frogs, after having cut off their feet, in a hemina of wine and washing of the mouth with the latter. Others tie them whole, on the jaw." "To strengthen unsteady teeth, rinse them with a decoction made by boiling ten frogs in three sextaries of vinegar, until the liquor is reduced to one-third," etc.

Guerini [1] says regarding the times of Pliny, "The diseases of the teeth were, in those days, most common: very often we find mention of loose teeth, and the medicines suited to make them firm again; from which we may deduce the great frequency of alveolar pyorrhea. Concerning the teeth, their affections, and the means of healing and preventing them, the strangest superstitions existed, and this not only among the common, but also among educated and learned people. The number of remedies reputed useful against diseases of the teeth was extraordinarily great. For the cleanliness of the mouth, for strengthening the teeth and gums, mouth washes of sundry kinds were adopted." Human urine seems also to have been a favorite remedy not only in Roman times but in succeeding centuries. In a poem by Catullus, reference to this custom is made in the following words, "Now, O Celtiberian, in thy Celtiberian land, each is accustomed, with the water he has himself emitted, to rub his teeth and gums. Wherefore the cleaner are thy teeth, the more surely stale dost thou accuse

thyself of having drunk." Guerini also states that "from Strabo we learn that the Cantabri and other peoples of Spain used to clean their teeth and sometimes to even wash their face not with fresh but with old urine, which, so it seems, was kept for the purpose in suitable cisterns."

THE MIDDLE AGES

From the days of Pliny, down through the Middle Ages, references to "loose teeth" and their treatment are made with increasing frequency, such references occasionally having a decidedly modern note. One such will be found in the writings of Paul of Ægina, a Roman of the seventh century, who says that "if a tooth projects above the level of the others, the protruding part must be removed with a file." Thus it will be seen that "traumatic occlusion" as represented by teeth that "project above the level of the others" was recognized in those far-off days and that the grinding of teeth by the use of files was recommended. This has a very modern sound at this time when so much is being said and written about excessive occlusal stress as an etiologic factor in periodontal diseases. As further evidence that "there is nothing new under the sun," Rhazis, a Persian of the ninth century, recommends astringent mouth washes for "loose teeth." Avicenna, a Persian of the tenth century, offers the following suggestion: "He who has a loosened tooth and desires to make it firm again must avoid using it in mastication, must not touch it with the fingers nor move it with the tongue." He also advises that they be "filed."

One of the great surgeons of the Middle Ages was Guy de Chauliac, who was born in a little village near Auvergne about 1300. It was his belief that "looseness of the teeth, which depends on dryness or want of nutrition as in old and consumptive people, is incurable." He recommends

that "loose teeth be tied to healthy ones with a little gold chain," adding that such teeth "are serviceable for a long time." The celebrated French surgeon, Ambroise Paré (1517-1592), urges that teeth "that stand out above the level of the others" be reduced by filing and bound to adjoining teeth that are firm, adding that "they may entirely reacquire their original firmness." Friedrich Ruysch (1638-1731), a physician of Amsterdam who made himself famous by his anatomical preparations and methods of embalming, made the important observation that absorption of the alveolar process invariably follows tooth extraction and always accompanies the loosening of teeth, stating that it precedes the falling out of teeth and is the cause rather than the effect of their loss.

FAUCHARD AND HIS WRITINGS

Pierre Fauchard (1690-1761) has been referred to as the founder of modern scientific dentistry.[1] While this may be something of an exaggeration, as judged by present-day standards, his work at least was a great improvement over that which had gone before, and marked the beginning of a new era in dentistry. His celebrated work, "Le Chirurgien Dentiste," was published in 1728, and afterward went through two French editions. It was also translated into German in 1733. He enumerates the different dental operations of that day as follows: "Cleaning teeth, separating them, shortening them, removing the caries, cauterizing, stopping, straightening crooked teeth, steadying loose teeth, trepanning, simple drawing of teeth, replacing them in their own alveoli, transplanting them to another mouth, and finally substituting artificial teeth for those wanting." It will be seen by the foregoing that "steadying loose teeth" and "shortening them" played a part in the practice of two hundred years ago. He also refers to periodontoclasia

as a "kind of scurvy." This theory was also held by Jourdain, who referred to it in 1778 as an "unknown kind of scurvy."[3] One of the remedies which Fauchard gravely recommends "consists in rinsing the mouth every morning and also in the evening before going to bed with a few spoonfuls of one's own urine immediately after it has been emitted, always provided the individual is not ill. One is to hold it in the mouth for some time, and the practice ought to be continued. This remedy is good but undoubtedly not pleasant, except so far as it procures great relief. Some of those to whom I have recommended it have assured me that in this manner they were relieved of pain to which, up to then, they had continually been subject. It is rather difficult in the beginning to accustom one's self to it; but what would one not do to secure one's self-health and repose." At least one improvement in the treatment of "loose teeth" in the more than three thousand years that had elapsed since this famous remedy was being recommended by the Chinese will be seen in Fauchard's recommendation that one use his own and not his neighbor's urine. His statement that "little or no care as to the cleanliness of the teeth is ordinarily the cause of all the maladies that destroy them" is one of the earliest expressions in history of the importance of oral hygiene in the prevention of dental diseases.

MODERN EUROPEAN WRITERS

In "Natural History of the Teeth," written by John Hunter [2] in 1778, reference is made to the "wasting of the alveolar process" which he says "begins at the edge of the socket and gradually goes on to the root or bottom. The gum, which is supported by the alveolar process, loses its connection and recedes from the body of the tooth, in proportion as the socket is lost; in consequence of which, first

the neck, then more or less of the fang itself, becomes exposed—the gums generally become extremely diseased: and as they are separated from both the teeth and alveolar process there is a very considerable discharge of matter from those detached surfaces."

One of the earliest attempts at instrumentation was practiced by Kunstmann [9] and described by him in a small book published in 1772. The operation seems to have consisted chiefly in the removal of subgingival calculus by instruments of his own design, though he at times resorted to gum resection.

Serre [9] refers to the operation as "very well devised, but tedious, as it has to be repeated at different times." He then goes on to state that "if, instead, the entire pocket is cut away at once and the root cleansed, the incision will close readily. The ultimate good results of this operation are quite satisfactory: Kunstmann, however, has forgotten to speak of the retraction of the gingiva from the destroyed section of the pericementum." So far as the author knows, this is the first mention made of the treatment of periodontoclasia by instrumentation, and the first reference to gum resection. Fox, in his "Natural History and Diseases of the Human Teeth," published in 1814, describes two different diseases of the alveolar process; one in which there is "considerable secretion of pus, which oozes out from under the gums at the necks of the teeth" and another in which there is no discharge, but "instead of being an absorption of socket, it is a filling up or contraction at the bottom of it, by which it becomes shortened and the tooth is constantly pushed out."

In 1873 Magitot [9] described what is today known as periodontoclasia, as a slow, destructive inflammation of the pericementum and alveolar process, beginning at the gingivæ and progressing toward the apex of the tooth, result-

ing in its final loss. He expressed the belief that disturbances in nutrition were the chief etiologic factors; accumulations of tartar he regarded as of little importance from an etiologic standpoint, though he recommended its removal as essential to successful treatment.

As one reviews the writings of these more or less ancient authors, including also those of more modern times, he is impressed by the fact that "loose teeth" was one of the common maladies of all peoples; also by the fact that the treatment recommended was, except in the occasional use of the cautery, usually of a therapeutic nature. Many of their remedies were of the most absurd and fantastic character, but their real error was in thinking that treatment was therapeutic rather than surgical, an error which persists in the minds of not a few, even to the present. This is seen in the countless number of nostrums that are being recommended for use in the treatment of "pyorrhea." These may be less unpleasant than their forerunners, but whether they are any more efficacious is an open question.

AMERICAN DENTISTRY

When one comes to review the history of American dentistry of a hundred or more years ago, it will be found that the dentists of those days were face to face with the same problem of "loose teeth" that for more than two thousand years had aroused the interest of their forebears. The disease was usually referred to by them as "scurvy of the gums," and was generally regarded as incurable. Little attention was paid to it by any one. Loose teeth became looser and were finally lost. The well-to-do had some artificial teeth of a kind, while those less fortunate went toothless. In the *Columbian Magazine* of 1789 the following news item appeared: "A newly discovered Remedy for the Scurvy of the Gums. Take half a pint of red sage tea;

add a piece of allum the size of a large nutmeg, and as much bole ammoniac; of honey a table spoonful; the same of vinegar: Set it on a slow fire, until the allum is dissolved; wash the mouth often. If the teeth are loose, add more honey, vinegar and allum with port wine." It will be observed that reliance was still being placed on therapeutic methods.

Then the change came. A man was heard to declare that "scurvy of the gums" was a curable disease and that its treatment was surgical and not therapeutic. This was a long step in advance. Up till then, few had regarded it as curable and few, if any, had succeeded in curing it. "Steadying loose teeth," "shortening them," "filing them," etc., had perhaps delayed their loss, but had done no more; they finally went the way of all teeth.

JOHN M. RIGGS

The man to whom more than any other individual in this country is due the credit for this step forward was John M. Riggs, of Hartford, Connecticut, the father of modern periodontia and one of the great names in American dentistry. Riggs was born in Seymour, Connecticut, October 25, 1810.[5, 6] His family was desirous that he should study for the ministry, and he was accordingly matriculated at Trinity College, Hartford, where he was graduated in 1837. It is reported that at the commencement exercises, the baccalaureate sermon was preached by one of the bishops of the Church on the subject of the "Trinity." At its conclusion Riggs, who had become greatly agitated, went to the bishop and said: "I believe in one God and only one God. I do not believe in a bouquet of Gods, and I'll be damned if I'll ever preach any such doctrine." Thus the Church's loss was dentistry's gain. Riggs then took up the study of medicine, but after one year began the study of

dentistry under a preceptor, as was the custom in those days, and began practice in Hartford in 1840. He was a close friend of Horace Wells, also of Hartford, the discoverer of anesthesia. When on December 18, 1844, Wells saw an exhibition in that city of the properties of "laughing gas" he was so impressed with its possibilities as an obtundent of pain in surgical operations, that the following day he invited Riggs to his office, and requested him to extract one of his teeth while he, Wells, was under the influence of the gas.[5] This was done, Wells declaring that he had suffered no pain in the operation, thus giving to Riggs the distinction of having performed the first surgical operation on record under an anesthetic.

Very early in his career, he became interested in the treatment of "scurvy of the gums" (periodontoclasia). It was his belief that the various phenomena attending its progress from the earliest manifestations of gingivitis to the final exfoliation of the teeth themselves were all different stages of the same disease. These he classified into four types corresponding to the degree of advancement made. Riggs seems to have written but few articles on the subject. One of these, entitled "Suppurative Inflammation and Absorption of the Gums," [10] appeared in "Johnston's Dental Miscellany" in 1878. He was, however, a frequent attendant at dental society meetings. At one of these held at Northampton, Massachusetts, in 1867, he gave a clinic at which he showed his methods of treatment, consisting of a rather heroic subgingival curettement, and exhibited patients he had previously treated.[7] At this meeting he stated that he had been treating cases in this way for twenty-five years, and that the majority of them had been successful. One year later, this same organization, the Connecticut Valley Dental Association, commemorated the event by a set of resolutions in which he is credited with

"originating and first publicly describing a method of treatment for the cure of inflammation of the gums and alveolar process, or so-called scurvy of the gums." [6]

In a personal letter written by Dr. Riggs to one of his patients, dated April 23, 1885, and given to the author by Dr. J. Edward Line, of Rochester, New York, he says: "The treatment originated with me and, owing to its difficulty of application, meets with great opposition. Physicians know very little of the disease and dentists less. They are not to be censured, for it is fifty years ahead of the age." In 1876 the Baltimore Dental College conferred upon him the honorary degree of Doctor of Dental Surgery in recognition of his outstanding achievements in the field of periodontology.[6] For several years he was clinical instructor at the Harvard Dental School. In 1881 he attended the meeting of the International Medical Congress in London where he was received with great respect. He died in Hartford, November 11, 1885, at the age of seventy-five. In very truth, he may be called the "Father of Periodontology."

HISTORY OF THE TERM "PYORRHEA ALVEOLARIS"

At the meeting of the American Dental Association held in Chicago in August, 1877, Dr. F. H. Rehwinkel read a paper entitled "Pyorrhea Alveolaris," and has been given credit for originating the term, it being the first time that it had been used in this country. The term had been in rather general use among European writers prior to its use by Rehwinkel, a fact of which he was undoubtedly aware.[5] Rehwinkel was born in Germany, June 15, 1825. After graduating in medicine, he emigrated to this country in 1848 or 1849 and became interested in the practice of dentistry. He subsequently took up its study at the Baltimore College of Dental Surgery, from which institution he was graduated in 1854. From that time until his death in 1889

he practiced dentistry at Chillicothe, Ohio. His chief claim to distinction in periodontology is his introduction into this country of the term "pyorrhea alveolaris." There is nothing in the record to indicate that he was especially interested in this field, though he speaks of having used Riggs' instruments with some degree of success. He consistently maintained that "pyorrhea" was not a purely local disease.[9]

WILLIAM J. YOUNGER

Among the most worthy of the early successors of Riggs was William J. Younger, who was born of British parents in Chile in 1838. At the age of ten he was brought by his parents to San Francisco, where he was later graduated in medicine. After practicing medicine for several years he took up the study and practice of dentistry, specializing more or less in what afterward came to be known as periodontia. In 1897 he removed from San Francisco to Chicago and from there in 1900 to Paris, where he continued in active practice up to the time of his death in 1920. There can be no doubt that he was a clinician of more than ordinary ability, even when judged by present-day standards. Like Riggs, he depended upon instrumentation rather than therapeutics, though his technique was more refined and his results more satisfactory. He devised his own instruments, many of which have never been improved upon. He was insistent in claiming that the *sine qua non* of successful treatment was *complete* removal of all calculary deposits and septic material from the root surface of all pockets.

That, however, which distinguishes him in the field of periodontology is that he was the first to observe and call attention to the fact that obliteration of pocket through reattachment of the surrounding vascular tissues followed correct treatment. In 1892, seven years after the death of

Riggs, he declared, "I am satisfied that attachment takes place in these cases, otherwise the teeth could not become so firm and the gums and tissues of the socket cling so to the tissues of the root. It is therefore an easy matter for direct union to be established between these healthy, living tissues when the partition of calculus is thoroughly removed from the root, and a granulating surface produced in the environing tissues of the gingivus." [5] But as was the case in the days of Riggs, little attention was paid to his teachings by the rank and file of the profession. No one, apparently, took the trouble to verify his oft-repeated statements regarding attachment, the foremost students of the histopathology of the periodontal tissues among his contemporaries claiming that suppurative detachments of the pericementum were permanent detachments.

A PERIOD OF TRANSITION

Following the death of Riggs and his immediate successors, periodontia passed through a stage of speculation and empiricism. Theories of all kinds flourished and found many adherents. There were those who believed that "pyorrhea" was an hereditary disease; that it was the local expression of systemic disease; that it was a form of catarrh; that it was infectious; that it was caused by uric acid, etc.[5] Ineffectual attempts at local treatment were occasionally made, but dependence was largely placed on drugs, acids, and certain antiseptics. The profession as a whole paid little attention to its treatment, with the result that, in most cases of periodontoclasia, it was only a question of time when the teeth became hopelessly involved and were extracted. Here and there a few had caught the vision of Riggs and continued his claims that "pyorrhea" could be cured and that its treatment was by instrumentation. One whose name is worthy of especial mention in this con-

nection is that of Dr. R. B. Adair of Atlanta, Georgia, who began the practice of dentistry in 1868 and is still in active practice. For more than sixty years Dr. Adair has consistently maintained the correctness of the principles laid down by Riggs, thus making him one of the beacon lights in the early history of periodontology.

Fads of all kinds have flourished in more recent days. With the introduction of cataphoresis great hopes were entertained that it would prove of value in the treatment of periodontoclasia. The same was true of the Roentgen ray. Both, however, proved a disappointment. Vaccine therapy was regarded as another "sure cure." Its application to the treatment of periodontoclasia was based on the part which bacteria play in its etiology. But since these organisms were not specific, the use of vaccines was illogical and proved to be ineffective. Their use in this country has been practically abandoned. Another fad was the so-called emetine treatment. The presence of endameba in periodontal pockets led to the conclusion that they were the active cause of periodontoclasia, and that their elimination by the use of an amebicide would be its logical treatment. Physicians, impressed by these claims, undertook the emetine treatment of periodontoclasia, as did many dentists. Lengthy articles appeared in the medical press and even books were written on the subject by medical men. As might be expected by anyone at all familiar with its etiology and pathology, failure was the result of such treatment. The lessons taught by Riggs and his successors had not yet been learned.

THE AMERICAN ACADEMY OF PERIODONTOLOGY

Meanwhile there had been growing up in this country a small group of dentists who had mastered the principles laid down by Riggs and his followers, and who had come

to be known as "pyorrhea specialists." They were all general practitioners, but the demands made upon their time was such, in some instances, that specialization became necessary, though, as a rule, it was carried on as a part of general practice: probably not a dozen men in the United States practiced it as a specialty. Periodontia as it is known today did not exist, and played little or no part in the practice of the average dentist. While the art of dentistry continued to advance by leaps and bounds, periodontia was almost wholly neglected. Mechanically perfect restorations were applied to teeth, the supporting tissues of which were involved in diseases that were bound to result in the loss of both restoration and tooth, with apparently no thought of the inconsistency involved in such practice. Unwittingly, the cart had been placed before the horse.

Periodontia as a specialty is a product of the present and may be said to have had its origin with the formation of the American Academy of Periodontology. This was an attempt on the part of a few to bring together for mutual study and improvement those scattered throughout the United States and Canada, who were known to be interested in this branch of dental practice. To the late Dr. Gillette Hayden, of Columbus, Ohio, who may be said to have been the leading spirit, and Dr. Grace Rogers Spalding, of Birmington, Michigan, with the coöperation of a small group, notably Dr. John Oppie McCall, of New York, and Dr. J. W. Jungman, of Cleveland, is due the credit for the formation of this organization. A call was issued to meet in Cleveland, Ohio, February 21, 1914, at which time a constitution and set of by-laws were drafted. These were submitted to a meeting held in Cleveland on May 23d of the same year, at which time an organization was effected and Dr. Austin F. James, of Chicago, elected president. This organization has met annually since its formation and has done much to place periodontology on a scientific basis.

Subsequently, periodontia was joined with orthodontia and made a section of the American Dental Association, which relationship was profitably continued until 1928. At that time, because of the rapid growth which each specialty had made, they were given individual representation in the sections of the Association.

From the foregoing it will be seen that the need for periodontia has existed for more than three thousand years, and still exists. How these needs are to be met, and the steps that must be taken to accomplish them, will be outlined in the following pages. It should be said, however, in this connection that there are still unsolved problems in periodontia, the solution of which are essential to complete success in the knowledge and treatment of periodontal diseases. While this is true, it is also true that a large measure of success will attend the intelligent application of present-day knowledge to the practice of periodontology. It is to encourage such application by the general practitioner of dentistry that the following pages have been written.

REFERENCES

[1] Guerini, Vincenzo, 1909, "History of Dentistry," Lea & Febiger, Philadelphia, Pa.

[2] Hunter, John, 1778, "Natural History of the Teeth," London, England.

[3] Jourdain, A., 1851, "Diseases and Surgical Operations of the Mouth."

[4] Kanner, Leo, 1928, "Folklore of the Teeth," The Macmillan Company, New York.

[5] Merritt, Arthur, H., 1921, "A Brief History of Periodontology," *Journal of Dental Research,* vol. 3, p. cxlix.

[6] Merritt, Arthur H., 1920, "Periodontology with Special Reference to Periodontoclasia," *Journal of Dental Research,* vol. 2, p. 77.

[7] Merritt, Arthur H., 1926, "Periodontia: A Retrospect and a Forecast," *Dental Cosmos,* vol. 68, p. 347.

[8] Moodie, R. L., 1928, "Studies in Paleodontology," *Journal of American Dental Association,* vol. 15, p. 1826.

[9] Prinz, Hermann, 1928, "Diseases of the Soft Structures of the Teeth and Their Treatment," Lea & Febiger, Philadelphia, Pa.

[10] Riggs, John M., 1878, "Suppurative Inflammation and Absorption of the Gums," *Johnston's Dental Miscellany,* vol. 5, p. 306.

[11] Riggs, John M., 1882, "Pyorrhea Alveolaris," *Dental Cosmos,* vol. 24, p. 524.

CHAPTER II

THE SUPPORTING TISSUES OF THE TEETH

An intimate knowledge of the nature, function and structure of the tissues which support and hold the teeth in place is essential to the practice of periodontia. This subject has been admirably treated by such authors as Black, Box, Noyes and Orban, all of whose writings should be carefully studied. The question which naturally suggests itself to the student of periodontia is: Why is it that of all the tissues of the mouth, those of the periodontium are the most susceptible to disease? The answer is probably to be found in the nature of their structure and in their relation each to the other. It is a case of a hard tissue, unlike that of any other in the body, being joined to another and held in place by a soft tissue. A joint is thus created, which Nature, apparently recognizing as a vulnerable point, has tried to protect by the elongation of the gingivæ in such a way that there is normally no direct contact with the cervical attachment. When, however, the gingivæ are injured by disease, as under conditions of modern life they are prone to be, they lose their protective property, and injury to the deeper tissues follows as a natural sequence. When it is also remembered that these structures are subjected to great stress, that interference in function occurs more often here than anywhere else in the body, and that they are continually subject to bacterial invasion, it will not be difficult to understand why these tissues, above all others in the human mouth, are peculiarly liable to disease. Still another explanation is to be found in the nature of

the periodontal circulation, which is of the terminal rather than the anastomotic type. The periodontal tissues are in a sense end organs, and therefore more liable to circulatory disturbances. The result is to increase their susceptibility to disease.

THE GUMS

The gums are represented by that tissue which covers the alevolar process and hard palate and are continuous with the gingivæ. There is nothing on the surface to indicate any demarcation between the gums and gingivæ, and for that reason some recent writers have preferred to include them all under the one term of gingiva, subdividing it into marginal, cemental and alveolar gingiva. While this has its advantages, the older terminology will be used here as being less confusing. By the gums is meant that tissue which clothes the alveolar process to its crest, the gingival tissues being those which surround the individual teeth and project themselves above the alveolar border. The gum tissue is a dense, inelastic tissue, the fibers of which are closely interwoven with those of the alveolar periosteum which explains its rigidity, and explains also why it is that gum recession rarely keeps pace with alveolar absorption in cases of advanced periodontoclasia. It is this union of the two tissues which prevents the movement of one upon the other, and gives to the gums their characteristic hardness and immobility. To a slightly less degree this is also true of the gingivæ. These tissues, including the gingivæ, are covered by a strong squamous epithelium which is closely united with the tissues beneath. For this reason the surfaces of the gums and gingivæ are not easily injured. They are also highly insensitive, notwithstanding the fact that they are richly endowed with blood-vessels and nerves. One has only to compare the insensitiveness of these tissues

with those of the eye, in order to appreciate the difference in endowments as to sensation given to different tissues to effect physiological purposes. They can, however, be aroused to great sensitiveness, if provoked by some irritation, such as undue pressure, acute inflammation, etc. The principal function of both the gums and gingivæ is defensive. The gums, especially, are not subject to disease and heal readily when wounded.

THE GINGIVÆ

The gingivæ are among the most interesting and important of the tissues which surround and support the teeth. Among those of the periodontium, they stand, as it were, on the very firing line between health and disease. Their outstanding function is to protect the subjacent tissues against disease and injury. No tissues of the human body are more marvelously adapted to their purpose. In health they project themselves above the attachment at the bottom of the gingival crevice, entirely surrounding the teeth at this point, and hugging them so closely as to prevent the impaction of food into the gingival crevice. It is at the bottom of this crevice that the attachment takes place between the hard and soft tissues. Because of the vulnerability of this point, elaborate provision has been made to protect it. Regarding this, Gottleib says: "In ideal cases the pocket (crevicular) epithelium is hornified down to the bottom (of the gingival crevice). The opposite tooth surface is covered with horny cuticle. The depth of pocket (crevice) is very small. Thus we find, at the weakest point of our denture, an ideal protection against injury."[4] Unfortunately, under conditions of modern life, "ideal" conditions do not always prevail, and the crevicular surface of the free gingivæ may be less well protected. Occasionally the crevicular epithelium will be absent altogether at

certain points and imperfect juncture with the cementum is of frequent occurrence. For these reasons, the crevicular surface of the gingivæ may generally be regarded as being the weakest link in the protection of the subjacent tissues against disease. (Fig. 1.)

Normally, the cemento-enamel attachment is at no time subject to direct impact. It is not until after the gingivæ have been robbed of their protective function by disease that this attachment becomes involved, and access is had to the subjacent tissues Under the combined influence of irritation and bacterial invasion, the gingivæ become inflamed, and food which in health glided over their outer surfaces without injury is crowded into the gingival crevice, adding to the irritation and further lowering their resistance to infection.

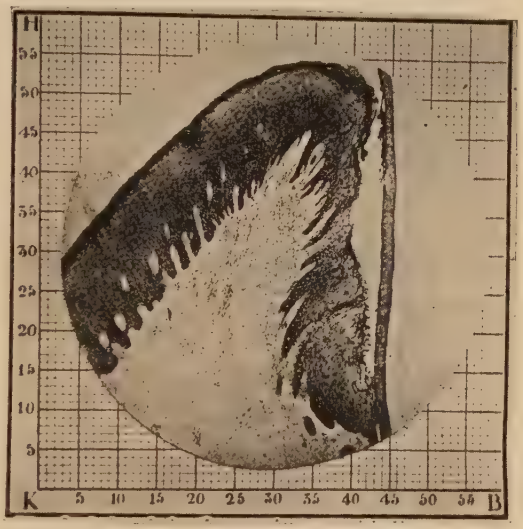


FIG. 1.—Gingiva showing break in the epithelium lining the cervicular surface. (From "Studies in Periodontal Pathology," by H. K. Box.)

While the general character of the gingivæ is not unlike that of the gums in its fibrous nature, it has a larger proportion of cellular elements and blood-vessels and is less dense. It is, moreover, practically immobile, and has a rubber-like tendency to bring its parts into apposition when cut, a fact which greatly facilitates healing. The gingivæ have an extremely rich blood supply, derived chiefly from

the pericemental and periosteal blood-vessels. The gingivæ may be divided into two parts: the body and the free gingivæ. The body consists of that part which rests upon the crest of the alveolar process, forming a soft-tissue extension of the alveolar process as far as the gingival line of the teeth. The free gingivæ are represented by those tissues that project themselves above the body of the gingivæ which encircles the cervical enamel. For convenience sake, the free gingivæ may be subdivided, according to their location, as septal gingivæ, lingual gingivæ, buccal gingivæ, etc.

In the prevention of all periodontal diseases, the gingivæ easily stand first in importance. To maintain them in health is the first duty of the dentist. All else in the practice of dentistry is secondary to this. Failure to do this may be, to the health of the mouth, what the loss of a horseshoe nail was to the army.

THE ALVEOLAR PROCESS

The alveolar process is that part of the jawbone which grows up and surrounds the roots of the teeth as they are erupted, thus forming the sockets in which they are held by the pericementum. It is composed of compact and cancellous bone, arranged in such a way as to best meet the stress imposed upon it by mastication. It serves no other function than that of supporting the teeth; extract them, and it disappears. Throughout its existence, it is continually being modified by environment, stress, exercise, etc. Noyes states that there is a "continual oscillation between formation and destruction by means of which it is perfectly adapted to its mechanical environment." [5] Vigorous use of the teeth and jaws in mastication tends to increase their strength and density. Conversely, failure to use the teeth and jaws makes for a frail and more easily broken-down alveolar process. It is generally known that the jaws

of primitive races were larger and more massive in structure than are those of modern peoples. This was largely due to the vigorous use given them in the mastication of coarse and more or less uncooked foods. Not only was the alveolar process increased in size and density in order to meet the strain imposed upon it, but its susceptibility to disease was likewise decreased—a fact which needs to be remembered in considering present-day problems in periodontia. No tissue is more subject to environmental influences. It will be found that the thickness and spacing of the trabeculæ in bone will vary with the intensity of the stress to which it is subjected, always being most dense in the regions of maximum stress. This, of course, depends upon its capacity for increased nutrition. Failure in modern life to vigorously use the teeth and jaws has brought about certain degenerative changes, represented by disuse atrophy of the alveolar process, malocclusion, dental caries, impacted and missing teeth, periodontal lesions, etc. Unfortunately, disuse lowers vitality, at the same time that it increases susceptibility to disease.

"The alveolar process is an outgrowth of a permanent portion of the skeletal structure, provoked and maintained by the existence of what are really dermal appendages and not true bones. It is a provisional structure, almost unique of its kind in adult life, which has apparently little independent vitality. This is in part due to mechanical causes, since its slender structure unsupported by the teeth offers little resistance to impacts from without, to which its situation renders it especially liable. There is more than this to be considered in reckoning the special vulnerability of the alveolus. It is protected by a thin layer of fleshy tissue only, which itself is peculiarly liable to injuries, thus exposing the bone to infection. There is, moreover, no cavity of the body that is more open to germs than the mouth, and

some of these that are considered especially virulent are its constant residents. Access of germs and accidental pathogenic factors to the alveolus is constantly afforded by carious teeth, the irritation of tartar deposits, etc. Its liability to disease is thus indefinitely increased. As it is not very resistant to morbid influences, the infections thus have free play, hence suppurative disease is more readily induced." [9]

THE CEMENTUM

The cementum is a specialized tissue that surrounds the roots of teeth, completely covering the dentine and frequently overlapping the gingival enamel. It is unlike any other tissue in the body. It is laid down by the pericementum in much the same way that subperiosteal bone, which it greatly resembles, is built by the periosteum. It is thickest in the periapical region and grows thinner as it approaches the cemento-enamel junction. Its thickness increases with age and under certain pathological conditions. It differs from bone in not being possessed of an Haversian system, though canals containing blood-vessels are not uncommon. It is a tissue that is continually undergoing changes, new layers being added as absorption occurs. Its function is to form an attachment for the connective-tissue fibers of the pericementum which hold the teeth in place, and aid in supporting the surrounding tissues.[1]

"If, for any reason, the tissues are detached from the surface of the root, they can only be reattached by the formation of a new layer of cementum on the surface of the root, which will embed the surrounding connective-tissue fibers. In order to accomplish this the tissues must lie in physiologic contact with the surface of the root, and the connective-tissue cells must be actively functional. That the tissues may be reattached to the surface of a root

is both theoretically possible and clinically demonstrable, but for it to occur, biological laws must be observed and the conditions are very difficult to control, especially with the old methods involving the excessive use of strong antiseptics. It is well to remember 'that a dentist can never cure a suppurating pocket along the side of a tooth root,' but if the conditions can be controlled the cells of the tissue may form a new layer of cementum, reattaching the tissues and so close the pocket. It is a biological problem, not a matter of drugs, except as they are a means of producing cellular reaction." [5]

THE PERICEMENTUM

The pericementum, sometimes called the peridental membrane, periodontal membrane, etc., is a tissue of great interest to the student of periodontia, for it is in it that some of the early manifestations of disease occur. Its function is to hold the teeth in place and in their normal relations to each other, at the same time admitting of a certain amount of motion. It is a very active tissue, being abundantly supplied with blood-vessels and nerves. Like all soft tissues, it is subject to inflammation and suppuration, but tends to heal rapidly once the cause of irritation is removed. The fibers of the pericementum, composed chiefly of white connective tissue, pass in various directions from the surface of the cementum, cross a small space, and are attached to the inner walls of the alveolus. Several groups pass into the gingivæ. They serve to hold the teeth in place and to maintain them in their normal occlusal relations. One of these groups of fibers, known as the gingival group, passes from the cementum and is distributed directly to the free gingivæ throughout their circumference. It performs the very important function of maintaining the gingivæ in their positions of close adaptation to the teeth,

in this way serving to protect the subjacent tissues against injury, especially the cemento-enamel attachment at the floor of the gingival crevice. Another group of fibers which, like the gingival group, is of great clinical interest is the transseptal group. These fibers pass across the septal space from one tooth to the other, to which they are attached, thus binding the teeth together without interruption, from third molar to third molar. The continual pull of these fibers has for its purpose the holding of the teeth together in their mesiodistal relationships, to the end that normal contact between the teeth may be maintained. It is not difficult to understand why it is, when disease involves the pericementum, and these groups of fibers are no longer able to function, that the teeth tend to drift from their normal relations to each other and to those of the opposing jaw.

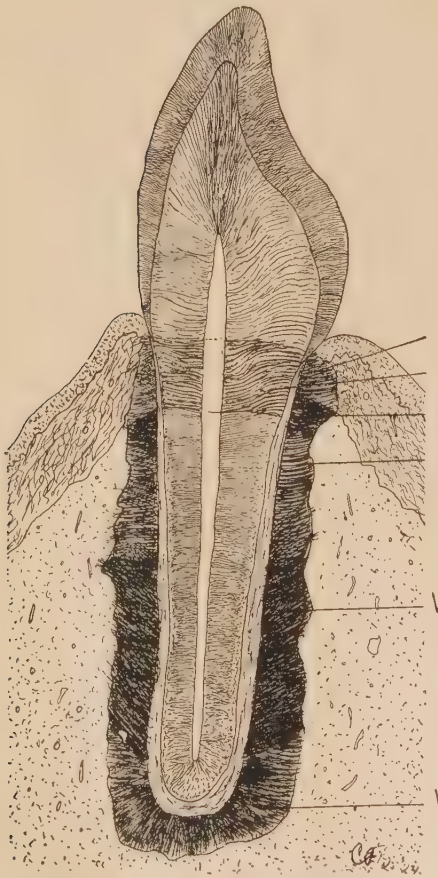


FIG. 2.—Classification of the six groups of the fibers of the pericementum. (After Black, Noyes and Bodecker.) I, Free gingival group; II, transseptal group; III, alveolar crest group; IV, horizontal group; V, oblique group; VI, apical group.

The groups of pericemental fibers are six in number: [1] The *gingival group,* the *transseptal group,* both of which have been referred to above; the *alveolar crest group,* consisting of those fibers that pass into the body of the gingivæ and are attached to the crest of the alveolar process; the *horizontal group,* made up of fibers that pass at right angles to the roots of the teeth and are attached to the inner walls of the alveolus a little below the crest; the *oblique group,* consisting of those fibers passing in an oblique direction occlusally, and the *apical group,* in which the fibers are attached about the apical portion of the root and radiate in fan-shaped bundles to the surrounding inner walls of the alveolus. The fibers of the first three groups all extend into the gingivæ, while the last three are all confined to the alveolus. They are all so arranged as to give the maximum support to the gingivæ and teeth. It is only under adverse conditions that they break down and fail in their highly important protective function. (Fig. 2.)

REFERENCES

[1] Black, G. V., 1915, "Special Dental Pathology," Medico-Dental Publishing Co., Chicago, Ill.

[2] Box, Harold K., 1924, "Studies in Periodontal Pathology," Bulletin No. 7, Canadian Dental Research Foundation.

[3] Box, Harold K., 1928, "Treatment of the Periodontal Pocket," University of Toronto Press, Toronto, Canada.

[4] Gottleib, B., 1927, "Tissue Changes in Pyorrhea, *Journal American Dental Association,* vol. 14, p. 2178.

[5] Noyes, F. B., 1915, "Dental Histology and Embryology," p. 157, Lea & Febiger, Philadelphia, Pa.

[6] Orban, B., 1928, "Dental Histology and Embryology," Rogers Printing Company, Chicago, Ill.

[7] Orban, B., 1929, "A Contribution to the Knowledge of the Physiologic Changes in the Periodontal Membrane," *Journal American Dental Association,* vol. 16, p. 405.

[8] Orban, B., 1929, "Contribution to the Histology of the Dental Pulp and Periodontal Membrane with Special Reference to the Cells of 'Defence' of these Tissues," *Journal American Dental Association,* vol. 16, p. 965.

[9] Talbot, E. S., 1899, "Interstitial Gingivitis," p. 96, S. S. White Company, Philadelphia, Pa.

CHAPTER III

THE DISEASES OF THE PERIODONTIUM AND THEIR DIAGNOSIS

In the study of diseases affecting the tissues of the periodontium, one is at once impressed with the fact that however much they may differ one from the other, they have one characteristic which is common to them all—viz., it is in the gingivæ that they first manifest themselves clinically. In other words, the gingivæ seem to be the door through which disease gains entrance to the periodontal tissues. It is probably not too much to say that, if the gingivæ could be maintained in health, it would be possible by that expedient alone to prevent the development of most of the diseases which involve the periodontium. This cannot be made too emphatic, for if it is in the gingivæ that disease gains entrance to the tissues of the periodontium, their preservation in health is of vital importance. Without healthy supporting tissues, teeth cannot long continue to function, no matter what else may be done for their preservation.

Another characteristic which they have in common is their tendency to break down and destroy the involved tissues. Beginning as a mild and often unnoticed inflammation of the gingivæ, they involve, by extension, the adjacent tissues, slowly destroying the pericementum and alveolar process, causing the gums to recede and the teeth to loosen. Their tendency is to create conditions that are, to a certain extent, irreparable, no matter what the treatment.

If, now, one look below the surface and study those

factors which are less obvious, it will be found that in one other respect they have a common characteristic, and that is in their etiology. They are all, with few exceptions to be noted later, dependent for their development and progress upon the presence of bacteria. These organisms, except in the case of Vincent's infection, are not specific. They represent the normal bacterial flora of the mouth, the chief offenders being the streptococcus and staphylococcus. Under conditions of lowered resistance, these organisms invade the tissues of the periodontium, and slowly break them down. So fundamental are these two factors of lowered resistance and bacterial invasion, that if either could be eliminated, there would be few if any periodontal diseases. All other factors such as oral sepsis, food impaction, occlusal stress, errors in diet, systemic diseases, etc., any or all of which may enter into a given case, are important as etiologic factors chiefly as they contribute to lowering resistance or increasing the bacterial flora. If in the study of periodontology this fact be kept clearly in mind, it will rob the etiology of periodontal diseases of much of its mystery.

TYPES OF DISEASE

The diseases that commonly affect the periodontium are not many in number, though each may assume a variety of expressions and may so blend with one another as to confuse diagnosis and make treatment more difficult. Complications may also arise, as when a periodontal abscess develops in the tissues about a tooth with a deep pocket, or when an acute periapical infection follows death of pulp in an advanced case of periodontoclasia, both manifesting similar symptoms, yet requiring dissimilar treatment. For these reasons, it is essential, in the practice of periodontia, that one be thoroughly familiar with these different mani-

festations of the diseases of the periodontium and their differential diagnosis, for in no other way can one hope to succeed in their treatment.

The most common of these diseases and the ones with which the practitioner is face to face in daily practice are the following:

1. Gingival recession; 2. Gingivitis; 3. Vincent's infection; 4. Periodontoclasia.

These, with their various manifestations and subdivisions, represent the group of periodontal lesions, with which every dentist should be familiar. Not only are they easily cured if recognized in their incipiency and properly treated, they are of all the diseases occurring in the mouth the most easily prevented. For these reasons every dentist should be so familiar with their manifold expressions as to be able to make a differential diagnosis, and take such steps as may be necessary to prevent their development before they have reached a stage where treatment is difficult, and cure uncertain or impossible. One should learn to regard every departure from normal as represented by certain types of gum recession, gingivitis, etc., as possible preliminary stages in that most destructive of all periodontal lesions, periodontoclasia. He should also keep clearly in mind that the most important factor in their prevention is a clean and intelligently cared-for mouth. Intelligent care includes not only such care as the patient may give toward keeping the mouth clean, but the regular and persevering coöperation of the family dentist, who should supplement and make more efficient the patient's personal efforts. Nor does the dentist's responsibility end here. He must assume the rôle of teacher, and properly instruct the patient in correct methods of tooth-brushing, which should include gum stimulation. Having taught him the way in which he should go, he should continually watch over him to see that he

perseveres in well-doing. In addition to such care, he should see to it that, where possible, malocclusion in children is given proper treatment; that abnormalities in proximal contacts are corrected; that lost teeth are restored in such a way as to maintain normal balance in occlusion, and that such attention be given to diet as to insure, as far as that is possible, the maximum of health to the periodontal tissues. In a word, the family dentist should be the guardian and preserver of the health of the mouth as a *whole*, instructing the patient in the important part which he must play in accomplishing this, and supplementing his efforts by the enlightened application of every principle of modern dentistry. Thus it will be seen that periodontia, if it is to be successfully practiced, must comprehend the whole of dentistry: orthodontia for the establishment of normal occlusion; operative dentistry for the correction of abnormalities in tooth form; prosthodontia for the replacement of lost teeth without which physiologic function is impossible; and lastly, exodontia, for the removal of teeth hopelessly diseased. But more important than any or all of these which go to make up modern dentistry, and of surpassingly greater value to the patient, is prevention, a fact which will be frequently emphasized in the following pages.

TYPES OF GINGIVAL RECESSION

There are no less than three distinct clinical types of gingival recession, all of which are etiologically different and may be present in the mouth at one and the same time. In some instances this recession is due to shrinkage only, as is seen in old age and in cases of periodontoclasia where there is extensive bone absorption. In others, it is represented by an actual destruction of the gingivæ, as will be pointed out later. In both instances, the bone loss

antedated and was a factor in causing the gingival recession.

The gum and gingival tissues, it will be remembered, are superimposed upon and clothe the alveolar process. With its loss, it naturally follows that the gingivæ shrink back, and expose to a certain extent the surfaces of the roots. Because of the dense and inelastic nature of the gums and gingivæ, this recession of the soft tissues rarely keeps pace with the bone loss.

There are also cases of gingival recession due to actual destruction of these tissues. This is true in cases of tooth-brush abrasion, and in certain instances, in acute Vincent's infection, where the gingival tissues are destroyed without there having been any previous loss of the alveolar crests. This latter type is the more disfiguring, representing, as it usually does, greater and more irregular recession.

Whatever the cause of the recession, the results are always the same, namely; root exposure, with all that that connotes of unsightliness, hypersensitiveness and increased susceptibility to caries. It is a matter of such importance, and is so common in some form, that the dentist should be thoroughly familiar with it in its different manifestations, in order that he may successfully treat or prevent it. Unfortunately, it can in no case be cured in the sense of restoring the gingivæ to their original positions, though they can be restored to health.

The different clinical types of gingival recession as seen in practice are:

1. Atrophic; 2. Abrasive; 3. Inflammatory.

ATROPHIC GINGIVAL RECESSION

As its name implies, this type of recession is represented by an atrophy or shrinkage of the gingivæ, dependent upon a more or less coincident wasting away of the crest of the

alveolar process. (Figs. 3, 4.) Of all the types of recession it is the most recondite.[5] In its pure form it is characterized by a generalized recession of the gingivæ and alveolar process, which recession will be found to be at approximately the same level throughout the entire circumference of each individual tooth, at the same time affecting more or less uniformly all the teeth in the mouth. When uncomplicated, the gingivæ appear healthy, there is no gingival inflammation, and clinically no pocket formation. The teeth are not loose, notwithstanding the fact that the recession may have resulted in the loss of a considerable amount of bone support. Uncomplicated cases are not common.

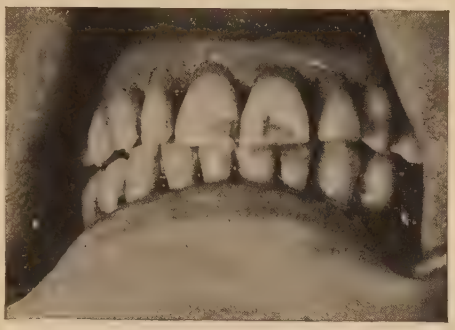


FIG. 3.—Atrophic gingival recession complicated by periodontoclasia.

FIG. 4.—Generalized alveolar atrophy and gum recession.

ETIOLOGY

A condition such as this is not uncommon in later life and by some is regarded as a normal physiological process. In those cases in which it is caused by senility, this is probably true. There is, however, another type of atrophic recession in which the etiology is not altogether clear. There are those [1] who believe that there is a type of periodontoclasia in which the initial lesion

is an atrophy of the alveolar process. Hopewell-Smith [6] asserts that pyorrhea "depends essentially upon an osseous lesion" and that these osseous changes are the result of a precocious senility. He states further that "in probably 80 per cent of individuals living in highly civilized communities, it is the normal condition for the alveolar processes to atrophy and shrink, for the gums to become thinner and degenerate, and for the roots of the teeth to become exposed.—Absorption of the alveolar process of the jaws is a common occurrence: It is the rule in man and the lower animals." With this Maurice Roy [4] is in entire agreement, but he is of the opinion that when it occurs in age, it is a "physiological process which could be due to vasoconstrictor phenomena." Fleischmann and Gottleib [1] call attention to an atrophic lesion of the alveolar process which they believe to be due to some constitutional factor. "If together with the atrophy of the bone there occurs a retraction of the gingiva, we have the picture of what is known in the literature as 'Atrophic Alveolaris.'" The causes of these atrophic changes in the alveolar process are obscure. Simonton [5] believes them to be due to disturbances in nutrition, which doubtless explains certain cases. When seen in persons well past middle age it is probably due, in some instances at least, to the wasting process of age. Whether it is but a preliminary stage in periodontoclasia, as is implied by the above-quoted authors, or a type of recession which may or may not have any etiologic relation to periodontoclasia, is a question which cannot be answered at this time. It would be logical to conclude that a condition which induces alveolar atrophy would be one that would also predispose to bacterial invasion and periodontoclasia, but it could hardly be called periodontoclasia until infection and pocket formation had occurred. Until then, it would be nothing more or less

than a condition of atrophy represented by bone loss and gingival recession. Whichever it may be, it should always be given serious consideration with a view to preventing further development.

TREATMENT

The most that can be accomplished by treatment is to prevent further recession. The bone that has been destroyed through absorption cannot be replaced, with the result that the receded gingivæ can never be made to occupy their original positions. Because of its atrophic nature, the increase of the blood supply through stimulation is the logical local treatment. This is best accomplished by the use of a toothbrush applied directly to the gums themselves, the technique of which will be described later. A high standard of mouth hygiene should be maintained at all times. The patient's efforts in this direction should be supplemented by prophylactic treatments at regular intervals. The vigorous use of the teeth and jaws in mastication should also be encouraged and such attention given to diet as may seem necessary. There can be no doubt that in certain cases of alveolar atrophy, diet deficiency is an important etiologic factor, a fact that needs to be kept in mind in studying all cases of atrophic gingival recession. The problem of diet in its possible relationship to diseases of the periodontium will also be considered later.

In the light of present knowledge, the most logical and effective treatment would seem to be the establishment of normal functional activity on the part of the jaws and teeth, regular prophylactic treatments, plus artificial stimulation by the intelligent use of the toothbrush. Such care should maintain the periodontal tissues in health and prevent to a certain extent, at least, any further recession.

RECESSION ON THE LINGUAL ROOTS OF UPPER MOLARS

Another type of recession, different, yet akin to the one under consideration, is that which is sometimes seen on the lingual roots of upper molars, usually the first, and occasionally seen on the second. (Figs. 5, 6.) In some instances the gingivæ and gum tissue will be found to have receded for more than half the length of the root, without any evidence of inflammation or pocket formation. This may be due to pressure atrophy, loss of the opposing tooth, or to

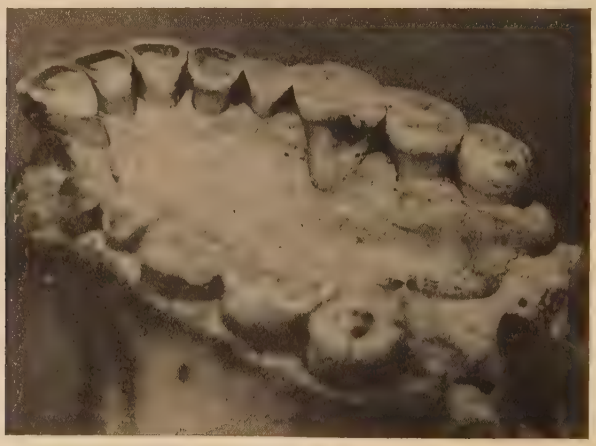


Fig. 5.—Exposure of root surfaces especially marked on lingual surface of first molar, due in large measure to occlusal wear. (Hirschfeld.)

outward spread of the lingual root, as a result of which there is only a very thin plate of alveolar bone interposed between it and the overlying soft tissues. In some cases the alveolar bone is entirely lacking over part of the root. It is desirable to study each case with a view to determining its cause. If due to extrusion of the tooth through loss of its opponent, replacement of the opposing tooth may be advisable. The condition is apparently non-pathologic.[2]

ABRASIVE GINGIVAL RECESSION

In this type, the recession is, with rare exceptions, limited to the buccolabial surfaces of the teeth. It is usually most marked on the left side, especially in the cuspid and bicuspid regions, though it may involve, to a less extent, the incisors and molars. It is rarely seen on third molars. The

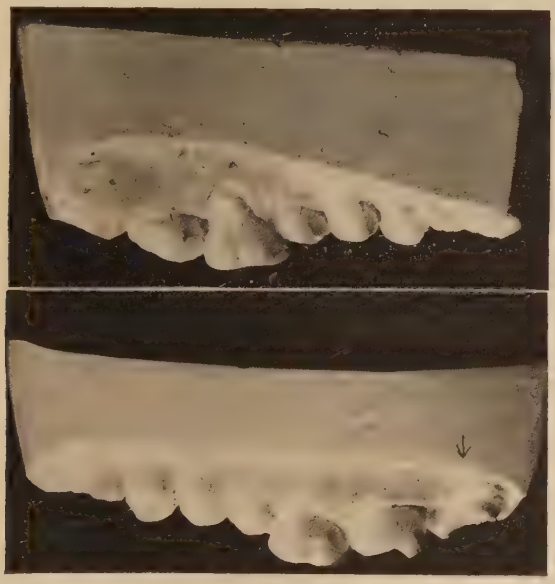


Fig. 6.—Gum recession on lingual root of upper first molar (above). Notwithstanding the fact that the tooth is extruded, there is no recession of the gingiva on the buccal side, as will be seen in model below. (After Hirschfeld.)

recession is often very extensive, sometimes to the extent of one-half of the length of the roots of the involved teeth. (Fig. 7.) Examination of the gum tissue (for in this type of recession the involved portions of the gingivæ have been destroyed) will reveal the fact that it is healthy and attached to the extreme limits of the receded area. It is generally hard, pink and insensitive. The affected teeth will be found to be less convex, often to the extent of being

almost flat over the receded areas. Sometimes they show deep, highly polished grooves, always more or less horizontal, and always presenting unmistakable signs of abrasion.

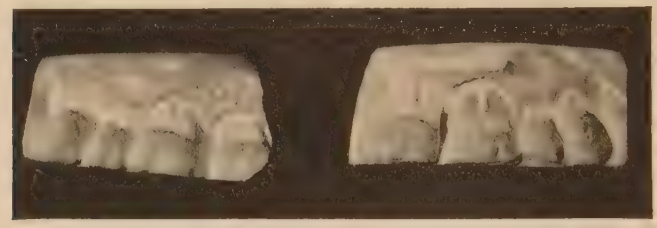


FIG. 7.—Abrasive gingival recession due to toothbrush. Patient age, 46. Note loss of tooth structure.

Scrutiny of the lingual gingivæ of such teeth will disclose the fact that they are in their normal positions unless complicated by other types of gum recession. (Figs. 8, 9.)

ETIOLOGY

Trauma by the toothbrush, plus in some instances highly abrasive dentifrices, explain this type of recession. A predisposing factor may be an abnormal buccolabial prominence of certain malposed teeth. In many cases the gingival portion of the root may never have been covered by alveolar process, having been protected only by the gum tissue, which under such conditions is less well nourished and therefore more susceptible to atrophic and abrasive influences. Cases are frequently met with in which the gingiva about a prominent cuspid, for example, is receded without

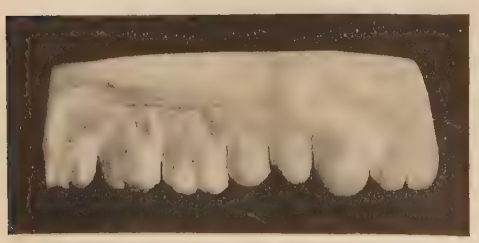


FIG. 8.—Abrasive gingival recession without tooth loss in patient of 25.

any abrasion of the root surface, showing how little friction may be necessary to cause recession of the gingivæ in such cases. Indeed, recession may have occurred without any friction whatever. Differential diagnosis can be made in the uncomplicated case from the fact that the recession is limited to the buccolabial surfaces, and that the exposed and abraded portions of tooth and root are usually less convex than normal. This latter is an important diagnostic feature.

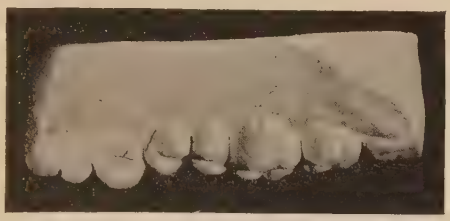


FIG. 9.—Lingual view of Fig. 8. Note absence of abrasive recession.

In advanced cases, there is usually considerable loss of tooth substance. This is not to be confused with erosion, which in some respects it resembles, and which in some cases may be a complicating factor. Erosion is due primarily to the solvent action of acids in more or less direct contact with the involved teeth, which are apt to be few in number as compared with those that may be affected by abrasion. (Fig. 10.) Moreover, erosion of itself never causes gingival recession. They may, however, both be present in the mouth at one and the same time and be so blended as to make differential diagnosis difficult.[3]

FIG. 10.—Typical case of erosion. Note absence of gingival recession and the fact that only three teeth are involved.

TREATMENT

There is no treatment that can be given that will restore the gingivæ to their normal positions, or the teeth to their original contours. Both represent tissues that have been

destroyed, and the power of re-creation is not possessed by anyone, not even the most skillful. Careful inquiry into the patient's habits of toothbrushing should be made, and correct methods taught, which must be learned and persisted in by the patient. Careful inquiry should also be made regarding the dentifrice being used. Some of those on the market are highly abrasive and capable of causing serious destruction of tooth substances, especially when the toothbrush is used in a horizontal direction. (Fig. 11.) By so doing one can do no more than prevent further recession, but that is worth while. Better yet, the dentist should observe his patients carefully in this respect, especially young patients, to the end that abrasive recession be prevented altogether.

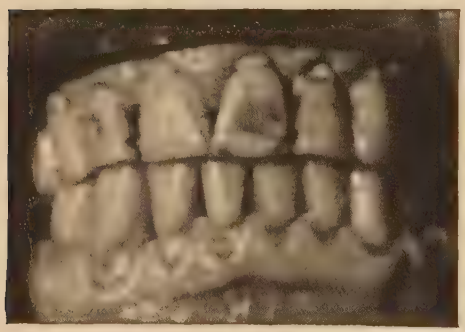


Fig. 11.—Abrasive gingival recession caused by horizontal toothbrushing.

It frequently happens that these abraded surfaces become highly sensitive, sometimes acutely so. Where they are not deep, and fillings are contraindicated, applications of formaldehyde, full strength, will often give permanent relief. It must, however, be used with caution, for if brought in contact with the soft tissues to any extent it will cause sloughing. Having protected these tissues with a mouth napkin, a very satisfactory and efficient method is to take a small orangewood stick, the point of which has been made wedge shape, and, dipping it in the formaldehyde, apply it directly to the sensitive area, meanwhile *rubbing* it into the tooth. This should be continued for several minutes. To insure against the use of too much formaldehyde, touch the wet point to a napkin to absorb

any excess, using only what will be absorbed by the moistened point. Usually one treatment will render the area insensitive to the touch of the toothbrush and less sensitive to temperature changes. Several applications over a period of a week or two can be made, if necessary, though one or two will usually suffice. The subject of desensitizing these areas will be taken up at greater length later. When the areas are deep, fillings are usually indicated.

INFLAMMATORY GINGIVAL RECESSION

This is a type of recession that is associated with, and dependent upon, the presence of some disease in the periodontium. It is a term not altogether satisfactory, but is used to indicate a type of recession that depends for its existence upon a disease process accompanied by inflammation, of which recession is merely one of the symptoms. Inflammation of the gingivæ, and loss of alveolar process are always present, and pocket formation and suppuration may be also. The whole condition is obviously one in which the recession is caused by a preëxisting disease, which makes its etiology clear. It cannot, therefore, be confused with either of the foregoing types. (Fig. 12.)

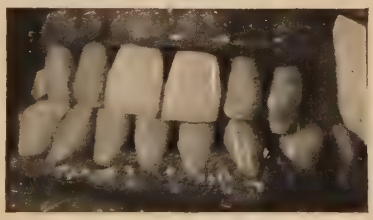


Fig. 12.—Type of inflammatory gingival recession caused by disease.

TREATMENT

To prevent further recession, it is necessary that the underlying disease receive such treatment as may be necessary to clear it up. What that treatment should be will, of course, depend upon its nature, whether it be Vincent's infection, gingivitis, or periodontoclasia. Their treatment

will be indicated later. In every case it is of first importance that a correct diagnosis be made.

REFERENCES

[1] Fleischmann and Gottleib, 1921, "A Contribution to the Histology and Pathogenesis of Pyorrhea Alveolaris," *Dental Cosmos,* vol. 63, p. 215.

[2] Hirschfeld, I., 1926, "Some Types of Periodontoclasia and the Diagnosis of their Incipient Symptoms," *Dental Items of Interest,* vol. 48, p. 836.

[3] McDonagh, A. J., 1919, "Periodontology, with Special Reference to Recession of the Gums," *Journal of Dental Research,* vol. 1, p. 423.

[4] Roy, Maurice, 1918, "The Pathogenesis and Prophylaxis of Pyorrhea Alveolaris," *Dental Cosmos,* vol. 60, p. 659.

[5] Simonton, F. V., 1927, "The Etiology of Parodontoclasia," *Journal American Dental Association,* vol. 14, p. 1767.

[6] Smith, A. Hopewell, 1911, "Pyorrhea Alveolaris. Its Pathohistology," *Dental Cosmos,* vol. 53, p. 397.

CHAPTER IV

GINGIVITIS

The gingivæ represent the outposts of periodontal defense. Their function is to protect against injury the deeper and more vulnerable tissues and especially the cemento-enamel attachment—the *locus minoris resistentiæ* in the periodontium. In health, they surround the cervical enamel and so fill the interstices between the teeth as to prevent the lodgment of food, in this way performing a most important function.[4] Because of their exposed position in the defensive program plus conditions of modern life, they are frequently the seat of disease. It is almost impossible to find a person in mid-life with gingivæ that are entirely healthy and performing their normal protective function. (Fig. 13.) The result is not only disease of the gingivæ, but what is more serious, disease of the underlying tissues also. In general it may be said that anything that irritates them interferes with their protective function.

Fig. 13.—Chronic gingivitis in a young patient.

Gingivitis, as its name implies, is an inflammation of the gingivæ. In its earliest beginnings, and often for a considerable period, clinically speaking, this is limited to the gingivæ, but unless recognized and relieved, it is usually only a question of time when, by extension, it involves adjacent tissues. This is not to say that every case of gingivitis ends in periodontoclasia, and perhaps not every

case of periodontoclasia was preceded by gingivitis, but it is probably true that both had their origin in something which interfered with the normal protective function of the gingivæ. In the uncomplicated type, there is usually no pain, no pus and no pockets. Redness, tendency to hemorrhage and eversion of the gingivæ are the usual symptoms. Recession may or may not be present.

ETIOLOGY

Anything that lowers the resistance of the gingivæ to infection is an actual or potential cause of disease.[4] Among these may be mentioned functional inactivity of the teeth and jaws in mastication, interference in circulation, systemic diseases, bad hygiene, food impaction, tartar, certain drugs, faulty restorations, pregnancy, etc. Under the disturbing influence of any of these factors, there may be interference in function and nutrition, followed by bacterial invasion. When this occurs, the door is open to all manner of complications.[6]

TREATMENT

Treatment consists in the recognition of the factors which entered into the case under consideration and their removal, together with the institution of a program of stimulation. Bad hygiene and indifferent care will be found to be present in most cases. Both must be corrected. Each gingival crevice should be explored throughout its circumference and its contents removed. This will consist of food débris, bacteria, and in some cases calculary deposits. This latter is a product of the blood serum and known as serumal calculus. In health, there is a normal exudate from the gingivæ which continually bathes the gingival crevices. Under conditions of irritation, its character is changed and a deposit of serumal calculus is formed which

attaches itself to that portion of the tooth surrounded by the gingivæ. As this in turn acts as an added irritant to the already inflamed and irritated gingivæ, a vicious circle is established that can only be broken up by operative interference. Since none of the contents of the gingival crevice is usually visible, it will be necessary to explore each, in order to insure its complete removal. It is not enough to clean the teeth, no matter how thoroughly this is done. It is not what is on the tooth, but what is beneath the gingivæ and in contact with them, that is the real source of irritation in these cases. This is the reason why prophylactic treatments, as given by the dental hygienist, are inadequate. She is not permitted by law to go beneath the free gingivæ which is the seat of trouble in these cases, nor is she equipped by training or experience to do so. For this reason, her work should be limited very largely to the treatment of children or to adults possessing a normal periodontium.

Small curettes, to be described later, are well adapted to the removal of the contents of the gingival crevice. Their use should be followed by a careful and painstaking cleansing of the teeth. Applications to the teeth and gums of tincture of aconite root and iodine, equal parts, will serve the triple purpose of disclosing stain, germicide and stimulant.

POSTOPERATIVE CARE

Having finished treatment, it is necessary to instruct the patient in what constitutes proper care on his part. Remembering that impaired circulation in the tissues of the periodontium and possible disturbances in nutrition underlie nearly all diseases within its borders, it is essential that steps be taken to correct these. Vigorous exercise of the teeth and jaws, by including in the diet foods requiring

thorough mastication, should be encouraged. Correct habits of toothbrushing (also to be described later) should be taught, with a view to stimulating by artificial means the circulation in the periodontium, to the end that resistance to infection be raised. Complete and permanent cure of gingivitis of local origin will follow such treatment when intelligently carried out.

Having in mind the fact that gingivitis is one of the most common diseases occurring in the mouth; that by neglect it may so involve the deeper tissues as to cause the loss of the teeth; that when limited to the gingivæ it is quickly and easily cured, it must be obvious that there is in the whole realm of dental practice no one thing more important than the early recognition and treatment of gingival inflammation.

METALLIC POISONING

There is a type of gingival inflammation, not as common as it once was, that is caused by metallic poisons. Chief among those causing such inflammatory reactions in the gingivæ are mercury, lead, bismuth, arsenic, copper and phosphorus. If for any reason these poisons find their way into the general circulation to any considerable extent, they produce marked reactions in the gingival tissues and pericementum, rendering them peculiarly susceptible to inflammation and infection. They seem to possess an affinity for the gingival tissues in which they interfere with circulation and nutrition, acting as irritant poisons. When in the past it was common practice to prescribe calomel (*mercurous chloride*) for pretty much all the ills to which the flesh was heir, salivation and gingival disturbance was of frequent occurrence. "The inhalation of fumes or dust containing mercury by workmen in mirror factories is also said to be a cause of chronic poisoning." [1] According to Tal-

bot,[13] workers in mercury mines are usually toothless by the time they have reached the age of thirty-five.

The blue line in the gingivæ, so characteristic of mercurial poisoning, is believed to be "due to venous congestion and to insoluble salts of mercury which become lodged in these tissues." [2] When the circulation is thus interfered with, there is local irritation by accumulation in the blood-vessels, plus a weakened general resistance due to disturbance of the eliminative organs. The logical result is degenerative changes of a more or less serious nature. There are those who believe that the mercury in the gingival circulation combines with the sulphides of the saliva or those formed as a result of food decomposition about the teeth, and that, as a result of this, there is produced an insoluble mercuric sulphide which accumulates in the gingival tissues.

FIG. 14.—Mercurial gingivitis, especially marked in lower jaw.

In the subacute stage of mercury poisoning, which is the type most apt to be seen in dental practice, there is an excessive flow of saliva; a dark bluish line is formed at the gingival border; the gums are highly inflamed; the gingivæ everted, and, in chronic cases, pockets are formed about the teeth, from which pus is discharged. (Fig. 14.)

The logical treatment in all such cases is the discontinuance of the administration of mercury, avoidance of exposure to same, and the institution of a high standard of mouth hygiene. This latter is of primary importance, for when this is done, reasonable amounts of mercury can be tolerated without injury to the gingival tissues. It is a matter of common observation in clinics where syphilis is

treated by the extensive use of mercury, that if the mouth be kept clean, relatively large quantities of mercury can be given without gingival disturbance. Also that the use of a dentifrice containing potassium chlorate is useful in such cases as a preventive of mercurial gingivitis. Bunting [2] says that "similar effects on the gingivæ are produced by other metallic poisons such as lead, bismuth, copper and phosphorus in the mouths of factory workers exposed to fumes or dust containing these metals. Painters and workers in lead frequently show evidences of plumbism which are expressed not only in disturbance of the nervous and digestive systems but also as discoloration and congestion of the marginal gingivæ. These, like the mercurial disturbances, are usually followed by severe gingival inflammations upon which infections are usually superimposed. Quite similar conditions may be produced by the therapeutic administration of large doses of bismuth in patients sensitized to that element. Workers in brass and copper, who have not been properly protected from the metallic dust, have disturbances of the gums with a greenish line about the gingivæ. Goadby ("Diseases of the Gums and Oral Mucous Membrane," Oxford Med. Pub., 1923, p. 252) found that in 12,000 yearly examinations of factory workers exposed to metallic dust, not a single instance of a blue line of metallic poisons occurred in persons having perfectly clean mouths. It would appear, therefore, that the degree of metallic deposition in the mouth is related to the state of oral hygiene that prevails." This latter observation is a matter of the utmost significance, for it emphasizes still further the exceedingly important part which oral hygiene plays in the prevention and treatment of all periodontal diseases.

Phosphorus is the most disastrous of all the metallic poisons in its effect upon the periodontal tissues. Fortu-

nately it is much less common than was the case when it was extensively used in the manufacture of matches and where the workers were not protected against its fumes. It produces more or less extensive necrosis of the alveolar process and gingival tissues, but unlike cases of lead, copper and mercury poisoning, it is usually localized in those regions of the periodontium which have been exposed to it directly. It is most apt to occur in cases in which there is gum recession, gingival inflammation, etc. It must be regarded as a local rather than a systemic poison. Healthy mouths, free from dental caries, and periodontal infections, are, as a rule, in no danger of phosphorus poisoning. Since the replacement of white phosphorus in the manufacture of matches with the less poisonous form, the sesquisulphide, aided by the institution of dental supervision of employees, phosphorus poisoning has become much less frequent. Its treatment, like that of mercurial poisoning, consists in the prevention of further exposure to the fumes of phosphorus, and good oral hygiene.

Arsenical necrosis is the most common form of metallic poisoning met with in dental practice, though it is much less frequently encountered than was the case when tooth devitalization was the chief "indoor sport" of the dental profession. Arsenic trioxide ($As_2 O_3$), the form most frequently used in dental practice, is a highly active protoplasmic poison, which, when brought into direct contact with any vascular tissue, produces a necrosis, the extent of which will depend upon the amount so used. Exceedingly small quantities, such as may escape from a tooth in which it has been sealed, will sometimes cause extensive necrosis of the periodontal tissues when brought into direct contact with them. Indeed, if not arrested, it may cause the loss of both alveolar process and tooth. Bunting [2] is on record as saying that "when it is hermetically sealed within the

tooth for the purpose of devitalizing the pulp, it produces an intense hyperemic reaction in that tissue which results in thrombosis and rapid death of pulp. Although the drug may produce moderate toxic effects on the periapical tissues, when properly applied, it seldom actually passes through the apical foramina of fully developed teeth." When, for any reason, tooth devitalization by arsenic is indicated, it is best accomplished by drilling a small cavity into healthy dentine, placing a *very small* amount of arsenic on the floor of the cavity so made, and sealing it with oxyphosphate of zinc. Applied in this way it usually renders devitalization painless. It should be allowed to remain twenty-four to forty-eight hours and may safely be allowed to remain longer if necessary. It is never wise to place it in a carious cavity or directly upon the pulp itself.

Treatment for arsenical necrosis consists in thoroughly removing all necrotic tissue and the local application of hydrated oxide of iron which, it is said, will neutralize the effect of arsenic on the tissues. This will not be necessary unless some portion of the arsenic remains. A very satisfactory remedy for the treatment of arsenical poisoning of the periodontal tissues, following its use in tooth devitalization, is olive oil, sometimes referred to as sweet oil. Having removed the necrotic tissue with a spoon excavator, loosely pack cotton saturated with the oil into the affected area. It relieves pain and promotes healing.

PROLIFERATIVE GINGIVITIS OF PREGNANCY

Although pregnancy has been enumerated among the possible etiologic factors in gingival irritation, there is probably no type of gingivitis that can be said to be peculiar to and found only in pregnancy. (Fig. 15.)

When, however, gingivitis is present under conditions of pregnancy, especially when accompanied by oral sepsis, it

occasionally assumes a hypertrophic form in which tumor-like masses are to be observed. (Fig. 16.) If not treated, these tend to increase in size during the term of pregnancy and to diminish or disappear altogether after delivery.[9] They attract attention because of their size and because of the generalized and exaggerated type of gingivitis that usually accompanies them. Because of their inflammatory nature, they are not to be classed as tumors, which in some instances they closely resemble. Care should be exercised in diagnosis not to confuse them with epuli. A differential diagnosis can usually be made from the fact that the patient is pregnant, and that in addition to the tumor-like growth there is usually present a more or less generalized gingivitis of an exaggerated type. Also since in some cases these growths show a disposition to recur with each pregnancy, a careful inquiry should be made of the patient regarding her condition in former pregnancies, if any, as a history of such gingival disturbances will naturally aid in diagnosis.

FIG. 15.—Hypertrophic gingivitis in a man; same type as seen in pregnancy, confirming opinion that there is no type characteristic of pregnancy. Lack of hygiene primary cause.

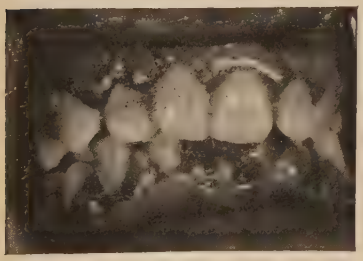


FIG. 16.—Hypertrophic gingivitis: patient pregnant. Gave history of always having had "bleeding gums," but had been worse since pregnancy.

TREATMENT

Treatment consists in removing from the mouth all sources of irritation and the institution of a high standard of oral hygiene, which should be maintained during the

entire term of pregnancy. In addition the gums should be regularly stimulated by the intelligent use of the toothbrush. By such care considerable reduction will take place in the hypertrophied gum tissues in which such growths are present. In those cases in which they do not disappear within a few months after delivery, they should be surgically removed and a microscopic examination made to determine their nature.[12]

The essential thing to be remembered, in connection with these cases, is that it is of prime importance that a high standard of mouth hygiene be observed and that in doing so, most if not all cases of proliferative gingivitis can be prevented altogether.

GINGIVITIS OF SYSTEMIC ORIGIN

There can be no doubt that, in certain cases, systemic disorders underlie, and to a considerable extent cause, gingival inflammations; a fact which should always be kept in mind in dealing with all periodontal lesions.

The gums and gingivæ, as already indicated,[13] are end organs, supplied by terminal arteries and possessed of a relatively deficient venous drainage. The result is that they are highly susceptible to any kind of nutritional disturbance that interferes with their peripheral arterioles. Especially is this true when the blood is freighted with the abnormal toxic products of a perverted metabolism, as it may be in such diseases as diabetes, Bright's disease, gout, non-diabetic acidosis, etc.[5, 14] There is, however, nothing in the gingival lesions caused by these or other metabolic disorders that, taken by themselves, are diagnostic of the causative disease.[3] That is, it is not possible to tell by the appearance of the gingivæ what is the nature of the underlying disease causing the gingival inflammation.

Bright's disease may cause a gingivitis that to all appearances is identical with that caused by diabetes.

Nor is it usually possible to determine, on examination, whether an existing gingivitis is of local or systemic origin. This is unfortunate and makes it necessary in every case to give such local treatment as would be effective in clearing it up if uncomplicated by a systemic background. Failing to get results from such treatment, one should suspect systemic complications and seek the coöperation of the family physician. This is of the utmost importance, for frequently these diseases, when still in their incipiency, and before there is any tangible evidence of their presence elsewhere, manifest themselves in the gingivæ. And it is in their early stages that treatment yields its most satisfactory results. For these reasons, and because a differential diagnosis is usually impossible, one should always be on the alert in dealing with these cases, remembering that what appears to be a simple gingivitis may, in fact, be the early symptom of some more or less obscure metabolic disorder which, if not discovered in its early stages, may reach a point where treatment of any kind is unavailing.

REFERENCES

[1] Brown, G. V. I., 1918, "Oral Diseases and Malformations," p. 331, Lea & Febiger, Philadelphia, Pa.

[2] Bunting, R. W., 1929, "Oral Pathology," pp. 326, 327, Lea & Febiger, Philadelphia, Pa.

[3] Croftan, A. C., 1911, "Gingival Manifestations of Certain Metabolic Disorders," *Dental Cosmos,* vol. 53, p. 1365.

[4] Ellis, G. R., 1925, "Gingivitis and its Treatment," *Dental Cosmos,* vol. 67, p. 446.

[5] Ersner, W., 1926, "Relation of Diabetes in Dental and Oral Surgery," *Dental Cosmos,* vol. 68, p. 279.

[6] Freisell, H. E., 1921, "Causes of Infection of the Gingivæ and their Treatment," *Journal American Dental Association,* vol. 8, p. 363.

[7] Hirsch, J, 1911, "Gynecology and Dentistry," *Dental Cosmos,* vol. 53, p. 1072.

[8] Kilmer, J. A., 1926, "The Chemotherapy of Gingivitis," *Dental Cosmos,* vol. 68, p. 354.

[9] Monash, S., 1926, "Proliferative Gingivitis of Pregnancy," *Dental Items of Interest,* vol. 48, p. 500.

[10] Pinard, A., 1877, "Gingivitis of Puerperal Women," *Dental Cosmos,* vol. 19, p. 327.

[11] Prinz, H., 1928, "Diseases of the Soft Structures of the Teeth and their Treatment," Lea & Febiger, Philadelphia, Pa.

[12] Schroff, J., 1926, "The Histopathology of Hypertrophic Gingivitis," *Dental Items of Interest,* vol. 48, p. 574.

[13] Talbot, E. S., 1899, "Interstitial Gingivitis," p. 93, S. S. White, Philadelphia, Pa.

[14] Williams, J. B., 1928, "Diabetic Periodontoclasia," *Journal American Dental Association,* vol. 15, p. 523.

[15] Zentler, A., 1912, "Oral Development in the Progeny Influenced by the Buccal Tissues During Pregnancy," *Dental Cosmos,* vol. 54, p. 1119.

CHAPTER V

VINCENT'S INFECTION

VINCENT'S infection is an infectious disease which usually involves the gingivæ though it may occasionally occur in other parts of the mouth, or as not infrequently happens, in the tonsils. When it occurs in the tonsils it is known as Vincent's angina.[7] In acute cases, and when not complicated by other periodontal lesions, it is characterized by a grayish-white slough along the border of the involved gingivæ, which, when removed, leaves a raw and highly sensitive surface. (Fig. 17.) The usual symptoms are pain, tendency to gingival hemorrhage, halitosis, bad taste, malaise, depression, temperature, etc. It occurs most often on the septal gingivæ, or about the gingivæ of partially erupted third molars; in fact, any place in the mouth where the gingivæ are irritated and so rendered less resistant to infection. Around the edges of bady fitted crowns or fillings is always a favorable point for its development. It is potentially dangerous, sometimes having a fatal termination.

FIG. 17.—Acute Vincent's infection, especially marked in gingivae of upper teeth.

ETIOLOGY

Vincent's infection is generally believed to be due to the pathogenic action of the fuso-spirallary group of organisms

which have been found to be present in practically all mouths. Recent investigations have shown that, in this association, the principal part is played by several varieties of spirochetes, the pathogenic action of which is increased by the presence of anærobic proteolytic bacilli belonging to the group of fusiform bacilli.[8] The organisms are usually referred to as the *Bacillus fusiformis* and the *Treponema vincenti,* though there is still considerable confusion in dental and medical literature regarding the correct nomenclature of these organisms. Referring to Bergey's "Manual of Determinative Bacteriology," Stafne says: "The classification submitted by Bergey in association with a committee from the Society of American Bacteriologists is at present accepted and recognized by that society. The two organisms given in their classification as being causative are identified as *Borrelia vincenti* and *Fusiformis dentium.*" [13a] Both are anærobic. He undertook a study of 250 routine cases to determine the approximate incidence of the organisms in the mouth and to observe the general condition of the oral cavity in relation to the bacteria present. He found the Spirochætaceæ to be present in 100 per cent of the cases examined. Spirochætaceæ and *Fusiformis dentium* were both present in about 75 per cent of adult cases even when there was no objective or subjective symptoms of Vincent's infection. The percentage was greater in those cases in which the mouth was unclean and pathologic conditions were present.[13a] This means that the presence in the mouth of the so-called Vincent's organism is not of itself diagnostic of Vincent's infection. This cannot be made too plain, for it is probable that to a certain extent these organisms are present in all mouths and cannot be dispossessed for any length of time. On this point Stafne, as quoted by Musburger, says: "One thing I haven't been able to do is to get rid of the organisms at any one time,

not even for a day. Some of the men who are making a study of it claim that they stop treatment only when they get a negative smear on three successive days. I don't know what methods they use in preparing their smears, but I have used a double check, using dark-field and several prepared smears (stained) at each sitting, and if I didn't find them in one, I did in the other." [9] Because of the uniformity with which these organisms are found in the mouth there are those who question their relationship to Vincent's infection. Kritschevsky and Séguin say: "We can affirm that ulcero-membranous angina (Vincent's infection) is not caused, as Vincent believed, by a specific spirochete, but by an association of different forms of buccal spirochetes." [8] It is their opinion that they cause disease only in those cases in which tissue vitality is subnormal, and then that the infection will be in direct relation to the virulence of the organisms. Greenberg is skeptical of there being any etiologic relationship, saying, "Until the spirochete and fusiform bacillus have produced Vincent's infection in susceptible animals, the cause of this disease must remain a matter for further research." [5] Meanwhile, the clinician must continue to regard the disease as an infection which, under certain conditions, may develop in any mouth, and treat it as such by methods which experience has shown to be highly successful. In all doubtful cases, smears should be made and when found to be positive by the presence of these organisms in large numbers, one can be reasonably certain of diagnosis, though as Ellis [4] points out, there is greater need for an observing clinician than there is for a bacteriologist. When in doubt, one is always justified in placing the patient on an anti-Vincent's treatment, for in so doing no mistake can be made. The predisposing factors in etiology are smoking, oral sepsis and gingival tissues that are being irritated from any cause;

such as crowns, bridges, overhanging fillings, partially erupted third molars, etc.

TREATMENT

Treatment consists in the establishment of a high standard of mouth hygiene and the use of germicides for the devitalization of the Vincent organisms. At the first treatment, except in very acute cases, the mouth and teeth should be cleaned as thoroughly as is consistent without injury to the soft tissues. This, it is realized, is contrary to the generally accepted teachings on this point, which are based on the principle that in cleaning the teeth a certain amount of injury is inevitable and that the infection will, under such circumstances, be carried deeper into the tissues. Every one is agreed that operative procedures of any kind should be postponed in all cases of Vincent's infection, especially when acute. But while this is true, there is no reason why the mouth and teeth should not be made as clean as possible. If anyone has any doubt on this point, let him give a careful prophylactic treatment to the next case of Vincent's infection that presents itself, limiting it to one-half of the mouth, and note the difference at the next visit. One such experience should convince the most skeptical.[9] This can and should be done, without any injury whatever to the tissues of the mouth. As a rule, most cases of Vincent's infection represent mouths in which a low standard of hygiene prevails. (Fig. 18.) To mechanically cleanse such a mouth is a most effective way

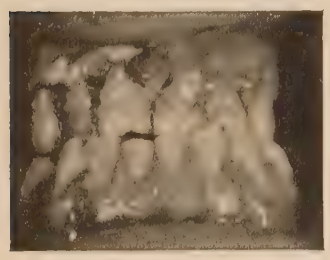


Fig. 18.—A long-standing periodontoclasia complicated by acute Vincent's infection. Note lack of care. First step in treatment is the establishment of good hygiene.

of reducing the Vincent organisms, raising tissue resistance and curing the disease. In acute cases this may be limited to simply polishing the exposed surfaces of the teeth, removing the grosser tartar, food débris, accumulated filth, etc., meanwhile spraying the mouth with a warm solution of hydrogen dioxide, two or three teaspoonfuls to four ounces of warm water. This should be prepared just before using. To the teeth, gingivæ and gums should then be applied a 5 per cent solution of chromic acid; a stronger solution is unnecessary. Several applications should be made at each visit. A very satisfactory way in which to make these applications, having first carefully scaled the teeth and sprayed out the mouth, is to apply the chromic acid to the teeth and gums, allowing it to remain while the teeth are being polished. It is then sprayed out and a similar application is made to the other jaw. At least two applications should be made in this way to each jaw, allowing it to remain for several minutes before spraying out. If desired, these applications may be alternated by the use of 5 or 10 per cent solution of neosalvarsan in glycerin. The important thing in connection with all local applications is that they be allowed to remain in contact with the affected tissues a sufficient length of time to accomplish their germicidal action. To apply them and immediately spray or wash out the mouth is not sufficient.

It should be remembered, in the treatment of Vincent's infection, that one is dealing with anærobic organisms, and that the logical therapeutic method is the use of some oxidizing agent. This is generally recognized, and most clinicians now have their pet formula. Of those oxygen-liberating drugs in most general use, chromic acid easily stands first as yielding the maximum of available oxygen, about 24 per cent. Potassium permanganate comes next,

with about 16 per cent and sodium perborate with 2 per cent.[3] The chief objection to sodium perborate as a mouth wash is that it is not very active in the liberation of oxygen. Indeed, "it is not active enough to be really considered a specific or to bring about a sudden termination of the disease."[6] As a mouth wash hydrogen dioxide is more satisfactory, as it liberates its oxygen far more readily. The use of proprietary preparations of unknown composition, so frequently advertised as possessing remarkable curative properties in the treatment of Vincent's infection, cannot be too strongly condemned. The sooner this is realized by the members of the profession the better it will be for them and their patients.

The following mouth wash may be given to the patient to be used in the intervals of treatment:

Hydrogen dioxide aquæ ..... 8 ounces
Mercury bichloride ..... 2 grains

Sig: Two or three teasponfuls in ½ glass of warm water and use frequently as a mouth wash, the oftener the better.

Smoking should be prohibited during treatment. In acute cases toothbrushing must be discontinued until healing takes place. Of the many treatments which have been recommended and tried out, the author has found that recommended above highly satisfactory.

Other preparations that have been recommended are the following:

Liquor potassii arsenitis ..... 2 ounces
Liquor antisepticus alk ..... 6 ounces

Sig: One teaspoonful in ½ glass of warm water and use frequently as a mouth wash.[2]

Salvarsan ..... 12 grains
Glycerin ..... ½ ounce

Sig: Apply direct to the affected parts.

Sodium chloride ..... ¼ teaspoonful
Powdered borax ..... ½ teaspoonful
Hydrogen dioxide ..... ½ teaspoonful

Sig: Dissolve in large glass of *hot* water and use as mouth wash three or four times daily. (Make fresh each time.)

Potassium permanganate .... 14 grains
Aquæ dist. q.s. ..... 6 ounces

Sig: Tablespoonful to ¼ glass of warm water and use as a mouth wash every two hours.

In acute cases the patient should be seen every day or every other day until all symptoms have disappeared. The mouth wash that has been prescribed should be continued several times daily during treatment. The following tooth powder to be used each time the teeth are brushed will serve as an excellent prophylactic and may be continued indefinitely:

Sodium perborate ..... 9 drams
Calcium carbonate precip. ... 3 drams
Pulverized castile soap ..... 45 grains
Saccharin ..... 2 grains
Oil of wintergreen ..... 15 drops
Oil of peppermint ..... 1½ drop

Sig: Tooth powder—Use freely two or three times daily.

The patient should later, when the lesions have healed, be instructed in the most exact methods of mouth hygiene. Brushes that have been in use should be discarded and new ones suited to the patient's needs prescribed. Correct technique as to their use should be taught. When the above conditions are observed, the most gratifying results are obtained. Relief is almost immediate, and there are few cases that need to be seen more than half a dozen times, usually less. Immunity, however, is not conferred by having had the disease. Reinfection may occur, especially if the patient is a heavy smoker and neglects the proper care of his mouth.

CHRONIC VINCENT'S INFECTION

There is a form of Vincent's infection lacking in many of the symptoms of the acute type that is very much more common, and which is often incorrectly diagnosed.[7] This is the chronic or subacute type, in which there is no slough and no marked objective symptoms. (Fig. 19.) The patient may complain of bleeding gums, tender gums, or of a bad taste in the mouth. Usually these are the symptoms of which complaint is made. Any one of these may be absent in a given case, but no case can be diagnosed as a subacute Vincent's infection in which there are not some subjective symptoms. Lacking these, a diagnosis of gingivitis can usually be made, though an anti-Vincent's infection treatment can always be safely given and, when in doubt, should be given. Careful examination will reveal gingivæ, the crests of which are a bright red, though this will not be uniform throughout the mouth. These are usually septal gingivæ, with possibly only two or three in the entire mouth that are involved. On pressure, they bleed easily, much more so than is common in cases of gingivitis. (Fig. 20.) They are also more sensitive. If any doubt exists as to diagnosis, smears should be made

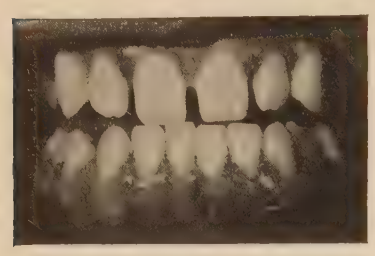


FIG. 19.—Sub-acute Vincent's infection. Note the septal gingivae between cuspid and lateral lower right side and from central to cuspid on left side. In maxilla, between lateral and central right side; lateral and cuspid left. Almost no destruction but several gingivae bright red and highly sensitive. History of recurrent attacks for several months. Excessive smoking chief predisposing factor. Hygiene above average.

FIG. 20.—Chronic Vincent's infection bordering on the acute. Also has advanced periodontoclasia. Bad hygiene a predisposing factor.

and examined. Treatment is the same as in acute cases and should be followed up until all symptoms disappear. Two or three treatments will usually suffice in these cases.

REFERENCES

[1] Buehler, A. G., 1920, "Vincent's Stomatitis and Associated Manifestations," *Dental Cosmos,* vol. 62, p. 847.

[2] Cahn, L. R., 1920, "Vincent's Gingivitis," *Dental Cosmos,* vol 62, p. 496.

[3] Coolidge, E. D., 1928, "Discussion," *Journal American Dental Association,* vol. 15, p. 2036.

[4] Ellis, G. R., 1925, "Gingivitis and its Treatment," *Dental Cosmos,* vol. 67, p. 446.

[5] Greenberg, S. and P., 1929, "The Incidence of Spirochete and Fusiform Bacillus in Gingival Inflammation and its Diagnostic Value," *Dental Cosmos,* vol. 71, p. 369

[6] Hardgrove, T. A., 1929, "Vincent's Infection from the Pathologic and Bacteriologic Point of View," *Journal American Dental Association,* vol. 15, p. 2034.

[7] Hirschfeld, I., 1928, "Vincent's Infection of the Mouth," *New York Medical Journal,* August 1, 1923.

[8] Kritschevsky, B., and Seguin, P., 1924, "The Unity of Spirochetoses of the Mouth," *Dental Cosmos,* vol. 66, pp. 511 and 622.

[9] Musburger, L E., 1928, "Vincent's Infection," *Dental Cosmos,* vol. 70, p. 520.

[10] Pilcher, Wm., 1925, "Vincent's Infection with Report of Cases," *Dental Cosmos,* vol. 67, p. 1064.

[11] Reichmann, F. J., 1926, "Vincent's Infection: Acute and Chronic," *Journal American Dental Association,* vol. 13, p. 1665.

[12] Smeltzer, J. L., 1921, "Diagnosis, Classification and Treatment of Vincent's Infection," *Journal American Dental Association,* vol. 8, p. 924.

[13] Stafne, E. C., 1928, "Spirochaetaceæ and Fusiformis Dentium in the Mouth," *Dental Cosmos,* vol. 70, p. 493.

[14] Vaughan, H. S., 1913, "Oral Infection by Vincent's Bacteria and its Importance to the Dental Practitioner," *Journal Allied Dental Societies,* vol. 8, p. 11.

CHAPTER VI

PERIODONTOCLASIA

Periodontoclasia, still known by the familiar though less appropriate name of pyorrhea alveolaris, has been the stumblingblock of dentistry from its earliest beginnings. Its complex and more or less obscure etiology, its various pathological manifestations, the exacting nature of its treatment, have all conspired to make of it a disease of mystery. This has been further complicated by the creation, in part, of a new nomenclature, which, though greatly needed, has temporarily added to the confusion. Notwithstanding all that has been said and written on the subject, and despite the clinical successes of the few, the rank and file of the profession have achieved little success in its treatment, and not a few are skeptical regarding its cure. The result is that the public, which has at its command excellent service in repairative dentistry, is often neglected in this important respect. This is not right. The health of the supporting tissues of the teeth is of paramount importance, for upon their health depends the very existence of the teeth themselves. To place the emphasis therefore on repairative dentistry, no matter how important, and to neglect that which should logically be the first concern of dentistry, is to neglect to do first things first. Except in advanced cases, or those complicated by tooth loss, treatment is not difficult and should form a part of the daily practice of every dentist, until such time as prevention shall have done its perfect work.

PATHOLOGY OF PERIODONTOCLASIA

Chief among the clinical symptoms of periodontoclasia are inflammatory reactions in the gingivæ and pericementum, absorption of the alveolar process, gingival recession, pocket formation, suppuration, tooth mobility, etc. Some of these symptoms may be absent altogether, and in incipi-

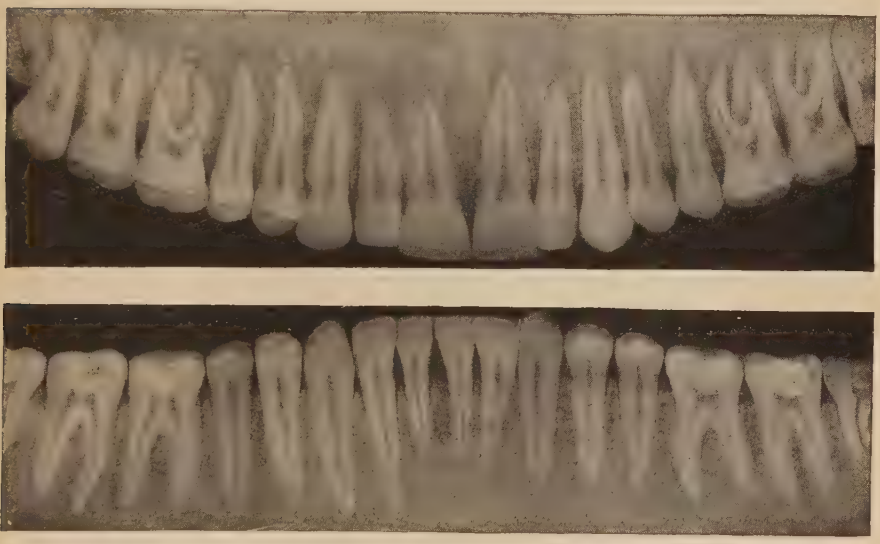


FIG. 21.—Panoramic radiographic view of a normal denture showing especially the height of the bony alveolar septi between the teeth, for comparison with other radiographs in which destruction of bone has taken place. (There is slight loss of bone between the lower incisors.) (From Black's Special Dental Pathology.)

ent cases usually are, but no case can be diagnosed as periodontoclasia in which to some extent, at least, impaired circulation, infection, alveolar absorption and pocket formation are not present. In making a differential diagnosis, pocket formation should be the deciding factor, for infection and alveolar absorption may, and frequently do, occur in the absence of periodontoclasia, though they may be but preliminary stages in its development. These pockets may be so slight in their incipiency as to make diagnosis uncer-

tain. It should be remembered, however, that pocket formation is a progressive phenomenon. Beginning as an inconspicuous lesion somewhere in the circumference of the gingival crevice, it advances circumferentially or vertically, slowly destroying the alveolar process and pericementum in its progress. (Figs. 21, 22.) This advance is usually made in a vertical direction; that is, toward the apex.

FIG. 22.—Panoramic radiographic view of the upper and lower jaw in a case of periodontoclasia of long standing. Note the amount and irregularity of bone destruction. (From Black's Special Dental Pathology.)

The reason for this is that somewhere in the circumference of the tooth, the conditions are less favorable to its progress, due generally to the better care given certain areas of the teeth and gums. Consequently it may involve only one surface of the tooth root, while another on the same tooth remains apparently normal—a fact which needs to be kept in mind in the prevention and treatment of these cases.

"HORIZONTAL AND VERTICAL PYORRHEA"

Occasionally reference is made to what is called "horizontal pyorrhea," in which it is asserted that the bone resorption is more or less uniform around the involved teeth; that shallow pocket formation and suppuration are the rule in such cases; that this type of "pyorrhea" far exceeds in frequency the type called "vertical pyorrhea" in which pocket formation is usually limited to one side of the tooth root and suppuration is not an early characteristic. This has not been the observation of the author. On the contrary, it has been his experience that bone resorption and pocket formation are much more prone to occur on those surfaces of the tooth root on which there are bacterial, calculary and filth accumulations, than on those that are more accessible to the scouring and stimulating influence of fibrous foods and toothbrush. For these reasons, pocket formation is most apt to occur in the proximal spaces and on the lingual surfaces of teeth, especially in the molar regions. There are, of course, exceptions to this rule, but even in such cases, it will usually be observed, if a careful study be made of the case, that some factor such as filth, occlusal stress, lack of functional activity, etc., is present to create at that point an area of lowered resistance. Usually in the hygiene of the mouth and teeth, certain areas are better cared for than others, with the perfectly natural result that such areas are more resistant to periodontal disease than are those less favorably situated. This is entirely logical; for if, as is very generally believed to be the case, functional inactivity and bacterial invasion are important etiologic factors in practically all periodontal diseases, then it must follow, as the night the day, that those areas less well cared for are more prone to disease. And since the phenomena of bone resorption and pocket formation are more or less progressive unless interfered with, it

is only natural that they should extend themselves vertically rather than circumferentially, inasmuch as it is in that direction that the resistance to infection and bone resorption is least effective.

Not all the teeth in the mouth are involved in this breaking-down process in the beginning. Indeed, it often happens that alveolar absorption and pocket formation will reach an advanced stage about certain teeth in a mouth, while other teeth in the same mouth continue unaffected and remain so throughout life. This would seem to indicate that in these cases at least (and they represent the majority of them), systemic conditions are not the controlling conditions. Also in those cases in which all the teeth are involved, it is a matter of common observation that their extraction is followed by a prompt and complete healing in the tissues of the periodontium, a further indication that there is something in the local conditions which explains its development. On this point Gottlieb says: "It is well known that, when we extract a pyorrhetic tooth, suppuration is checked at once. The inflammation was not in the tooth itself, and, by extraction, we have certainly not influenced the inflamed tissue or the micro-organisms. Yet the inflammation instantly disappears. By extraction there is only one thing which is certainly brought about, and that is the destruction of the pocket. The pocket consists of two walls, the gingivæ and the surface of the tooth. By removal of the tooth, the pocket has stopped forming. From these considerations, we may conclude that the presence of a pocket is the chief prerequisite for the existence of pyorrhea, the inflammation and the bacterial activity being of secondary importance." What actually happened by the extraction of the "pyorrhetic tooth" referred to by Gottlieb was the elimination from the periodontium of the conditions which were favorable to bacterial activity;

namely, the pocket and the dento-alveolar joint. For these reasons the extraction of the tooth profoundly influenced both the inflamed tissues and the micro-organisms. While it is safe to "conclude that the presence of a pocket is the chief prerequisite for the existence of pyorrhea," it cannot be said with the same assurance, and Gottlieb does not say so, that bacterial activity is of secondary importance in its relationship to the etiology of periodontoclasia and pocket formation. In all cases of "pyorrhea," not only do bacteria play an influential etiologic rôle, they are responsible also for its most characteristic symptom of pus formation. Without their presence in these cases, there could be no such thing as "pyorrhea." They are not, however, diagnostic of "pyorrhea," for bacterial activity may be a factor in other periodontal diseases, whereas pocket formation is the distinguishing characteristic of "pyorrhea."

FIG. 23.—Chronic gingivitis with incipient pocket formation. Radiogram of upper incisors show very little bone resorption.

FIG. 24.—Radiograms of upper incisors of Fig. 23, showing relatively little bone loss when compared with the hypertrophic gingivitis.

In the diagnosis of periodontoclasia, it should be kept clearly in mind, then, that pocket formation, no matter how slight, is its characteristic feature. Many cases that appear on casual examination to be a simple gingivitis will be found, when more carefully scrutinized, to be an incipient periodontoclasia. The disposition to regard as periodonto-

clasia only those cases in which the teeth are loose, suppuration abundant, and pocket formation deep, is a serious and, unfortunately, common mistake. (Figs. 23, 24.)

If the dentist who has not done so were to make a radiographic survey of a dozen adult patients, who on casual examination appeared to have a healthy mouth, he would probably be surprised at the amount of alveolar absorption which had taken place in many of them with very little visible evidence of disease. Indeed, the periodontist, who devotes all his time to these problems, is frequently surprised in just this way. On this point Hopewell-Smith says, "without any signs of gingivitis whatever, or the presence of tartar or pyorrhea, when the gum tissues are what one would call normal and healthy and the adjacent teeth fully functional and free from caries, decalcification or halisteresis of the free edge of the osseous socket may be beginning." [5] The lesson to be learned from this is that one cannot be too alert.

If one were to critically examine a typical case of periodontoclasia, it would be found that considerable bone loss has already taken place, and that this varies from tooth to tooth and about the different root surfaces of individual teeth. It would also be found that while there might be gingival recession, it is, as a rule, relatively little when compared with the bone loss. It is in this way that the pocket is created, for if gum recession kept pace with bone loss, there would be no pocket and no suppuration. Nor would there be the support to the teeth which these tissues give, following successful treatment.

THE HISTOPATHOLOGY OF PERIODONTOCLASIA

Antedating these clinical symptoms and in some instances more or less contemporary with them, are certain histopathologic changes occurring in the tissues of the periodon-

tium, far more subtle in their nature, which prepare the way for those grosser and more obvious symptoms with which all are familiar. These changes are described by Noyes as follows: "For many years the men who have studied most carefully the changes in the tissues occurring in the inflammations of the peridental membrane have been convinced that the conditions found were the reaction of the tissues to recurrent infections. . . . After studying many sections from a large number of teeth, it became apparent that the first positive changes occurred in the interfibrous tissue. That is, in the slight amount of connective tissue that normally fills the spaces between the principal fibres which pass in bundles from the cementum to the bone. Normally this tissue is small in amount and surrounds and accompanies the blood-vessels. . . . These spaces between the principal fibres become filled with inflammatory products which encroach more and more on the principal fibres pushing them aside. The blood-vessel walls in these areas also show inflammatory reaction. By the continuation of this process the normal arrangement of the tissue becomes profoundly altered." [3] Box calls the lesion referred to by Noyes, "rarefying pericementitis fibrosa," and describes it as a "chronic and progressive lesion of the pericementum which results in the rarefaction and destruction of alveolar bone. The process consists of the formation, in the pericementum, of a new tissue which, on the one hand, changes the normal arrangement of the pericemental structures, and, on the other, replaces the bone substance of the alveolar process. These changes in the pericementum and the alveolar bone are concomitant lesions. . . . Accompanied by osteoblasts and osteoclasts, the absorption of bone is seen to take place, with the formation of little bay-like depressions into the surface of the alveolar wall. Into these inlets, the new tissue extends

in finger-like projections along the blood-vessels, carrying inward reactions similar to those seen upon the surface, and producing within the bone an internal rarefaction." [1] (Fig. 25.) More recently Orban [4] has asserted that these changes are due to what he calls "wandering of the teeth" in a mesial direction. As a result of this "wandering" the fibers of the pericementum on the distal side of the teeth are stretched, while those on the mesial side are relaxed, causing resorption of the inner wall of the alveolus on the mesial side. This resorption of bone he believes is not the result primarily of changes in the pericementum, but the reverse; that is, the pericemental changes were caused by bone resorption. It is his opinion that the process is a more or less physiologic one. These changes, so important in the study of periodontia, are said by Noyes to be "caused by recurrent infections, producing intermittent, progressive destruction of the characteristic arrangements of the tissues." The natural outcome of this subtle breaking down of the tissues of the periodontium, in their ultimate results, is the formation of pockets, the loosening of teeth, and interference with their normal relations to each other and to the teeth of the opposing jaw. The rapidity with which

Fig. 25.—Rarefying pericementitis fibrosa showing the formation of nodular masses of new tissue in the pericementum with resultant concavities in the inner wall of the alveolar process. From "Studies in Periodontal Pathology," by H. K. Box.

these changes occur will depend upon many things, such as resistance to infection of the tissue cells of the periodontium; the regularity or irregularity of the teeth; the cleanliness or uncleanliness of the mouth; the vigor with which the jaws and teeth are used in mastication, etc. Progress is usually slow, years often elapsing between the genesis of disease and the ultimate loss of teeth. Meanwhile, the process of destruction goes on, and infection, always an actual or potential danger, continues as an ever-present factor.

REFERENCES

[1] Box, Harold K., 1924, "Studies in Periodontal Pathology," *Canadian Dental Research Foundation,* Bulletin No. 7, pp. 33 and 44.

[2] Gottleib, B., 1927, "Tissue Changes in Pyorrhea," *Journal American Dental Association,* vol. 14, p. 2201.

[3] Noyes, F. B., "Changes in the Peridental Membrane," *Journal of the American Dental Association,* vol. 7, pp. 125 and 126.

[4] Orban, B., 1929, "A Contribution to the Knowledge of the Physiologic Changes in the Periodontal Membrane," *Journal American Dental Association,* vol. 16, p. 405.

[5] Smith, A. Hopewell, 1911, "Pyorrhea Alveolaris—Its Interpretation," *Dental Cosmos,* vol. 53, p. 982.

CHAPTER VII

ETIOLOGY OF PERIODONTOCLASIA

The etiology of periodontoclasia is complex in the sense that there are always several factors entering into each case which vary from case to case. As a further complication, some of these etiologic factors are at present only partially understood and therefore not always recognized. And as if to make "confusion worse confounded" these multiple factors are frequently so commingled as to make it exceedingly difficult, if not impossible in some instances, to correctly appraise their relative importance. For these reasons, no one can afford to be dogmatic in any consideration of the subject, inasmuch as some of the theories now held and believed to be correct may be found to be erroneous in the light of further investigations.

In setting forth the various causes of periodontoclasia as they are at present understood, an effort will be made to present the more or less generally accepted theories, with a view to giving as clear an outline as is possible of all those factors which have to do with the etiology of those retrograde changes occurring in the periodontal tissues as a result of the admixture of local and systemic factors.

CLASSIFICATION

For convenience sake the etiologic factors in periodontoclasia may be divided into three groups as follows: First, those having a systemic origin; second, those which are purely local; third, those either systemic or local, which underlie and play an important part in all cases. Unfortu-

nately for the clinician, it is not always possible to differentiate between these factors in a given case, but he should be encouraged by the fact that the elimination of the factors in groups two and three, which are more easily recognized, will in most cases yield very satisfactory results.

SYSTEMIC FACTORS

There is slowly accumulating evidence pointing to the fact that there is a type of periodontoclasia in which the primary etiologic factor is systemic in origin. Regarding this Fleischmann and Gottleib [1] state that atrophy of the alveolar process, due to some constitutional disturbance, "is the causal and primary factor in the etiology of pyorrhea alveolaris," and that such atrophy can, and does, occur without any inflammatory reaction in the gingival tissues. While admitting that occasionally local inflammatory reactions in the gingivæ may by extension involve the alveolar process, and so act as the primary cause of periodontoclasia, they regard such cases as the exception and not the rule and therefore without great significance. This is concurred in by Hopewell-Smith, who asserts that pyorrhea alveolaris "is essentially dependent upon an osseous lesion, *an atrophy of the bone,* which in the thinnest parts causes the cervical margins of the teeth to become denuded through the halisteresis and osteoclastic absorption." [2] Simonton, who refers to this type as "periodontoclasia obscura," says it is characterized by an atrophy of the alveolar process in which the inflammation, if present, is secondary or sequential. He believes, however, that such cases represent only one type of periodontoclasia, and are not, as some appear to believe, typical of all cases. "I cannot agree with those authors who believe that all 'pyorrhea' begins as a marginal atrophy of the bone, for there is much evidence of a true inflammatory type in which the loss of bone is sec-

ondary." [8] Both Simonton and Gottlieb are agreed that whatever may be the systemic factor responsible for the bone atrophy, it is not due to constitutional diseases. Regarding this Simonton says, "Confirmatory of the evidence submitted above to the effect that alveolar atrophy is nutritional in origin and not connected with systemic disease (compare Gottlieb) is the material shown from a series of post-mortems and biopsies. The bone was found to be architecturally and biologically good (except as involved secondarily by advanced inflammatory cases) in tissue from a healthy American Indian, a tuberculous Mexican, a tuberculous Englishman, an aged and syphilitic Chinaman and a very sick boy of thirteen years." [8]

It is his belief that these retrograde changes are due to some disturbance in nutrition. In confirmation of this, he refers to results obtained by him in experiments on dogs in which a diet composed of bread, meat, milk, butter fat, orange juice, etc., and containing an excess of basic ions through the addition of sodium carbonate, produced marked changes in the alveolar process without any obvious change in the long bones. He refers also to the investigations of Howe, Grieves and Jones, in support of his belief that the atrophic changes occurring in the alveolar process, as observed by Gottlieb and others, are caused by some disturbance in nutrition, possibly to alteration in the acid-base balance. His conclusions may be summed up as follows: *First,* that the retrograde changes occurring in the alveolar process of experimental dogs fed on a diet known to produce bone degeneration, would indicate that, in certain cases of periodontoclasia, nutrition is an important etiologic factor. *Second,* that the microscopic changes seen in the alveolar process of these dogs closely resembles the "diffuse atrophy" observed by Gottlieb as occurring in man under certain conditions. *Third,* that the fact that these

pathologic changes occurred in animals having the appearance of being well nourished, is in harmony with the occurrence of periodontoclasia in human beings, who appear to be in perfect health.[8]

As a result of these investigations, one is justified in believing that there is a type of disease involving the alveolar process in which the primary cause is of systemic origin, probably due to some nutritional abnormality. Whether all such cases progress to the point of inflammatory reactions in the gingivæ, pocket formation, suppuration, etc., is uncertain. They certainly could not do so without the concurrent influence of local factors.

Clinical experience would seem to indicate that if these local factors could be eliminated altogether and a high standard of mouth hygiene maintained, supplemented by normal function of the periodontium, there would be little to fear from these atrophic changes in the alveolar process. And in those cases in which they did occur, if unattended by inflammation, suppuration and pocket formation, they could hardly be called "pyorrhea."

LOCAL ETIOLOGIC FACTORS IN PERIODONTOCLASIA

The etiologic factors in periodontoclasia which are purely local may be considered under four heads as follows: First, Functional inactivity. Second, Food impaction. Third, Oral sepsis. Fourth, Occlusal stress.

FUNCTIONAL INACTIVITY

By functional inactivity is meant the failure under conditions of civilization to give to the teeth and jaws the vigorous exercise in mastication which is essential to their normal development, and, what is equally important, their resistance to disease. If an examination be made of the teeth and jaws of primitive peoples it will be found that

as a rule they enjoyed a high degree of immunity to dental diseases. They retained their teeth throughout life free from caries, periodontoclasia or malocclusion. Small and malformed jaws, irregular teeth with frail osseous support were unknown among them. They were possessed of organs of mastication which were large and robust and capable of vigorous function. And that such function was given them in mastication is proved by the fact that, with adults, there was marked cuspal abrasion often to the extent of complete obliteration of all cusps. Like the biceps of the athlete, their jaws and teeth were highly developed, functionally active, and as a result more or less immune to disease. In addition, their diet consisted largely of raw, coarse foods, which made vigorous exercise necessary, at the same time that it furnished its full quota of nutritional value. Furthermore, they had ancestors who were free from periodontal disease, so that heredity may also have been a contributing factor to their immunity.

If this be contrasted with present-day conditions, one cannot fail to be impressed with the difference. Modern preparation of foods makes the use of jaws and teeth almost unnecessary; both are functionally inactive. The natural consequence is a diminution in size, frail alveolar bone, malocclusion, tooth impaction, undernourished tissues, and, most important of all, an increased susceptibility to disease through lowered resistance to infection.

And all this is largely accounted for by interference with function. When the cells of a tissue are undernourished, as they will be if the tissue of which they are the elements is functionally inactive, they lose to a certain extent their natural resistance to disease. And this is what occurs in the tissues of the periodontium. At birth the mouth is sterile, but almost from the first breath bacteria are present. It is impossible to sterilize the mouth and keep it

sterile; nor is it necessary. Under conditions of health, bacteria, even in large numbers, give little or no trouble. This is seen in the case of primitive man who, with a complete disregard of every principle of mouth hygiene, was not subject to periodontal disease. It is only when cell resistance is low that bacteria gain entrance to the tissues and produce disease. In childhood and youth, when the individual is active and the vital forces high, periodontoclasia is almost unknown. It is later in life, when the cumulative effects of functional inactivity, dietetic errors, bad hygiene, etc., make their appearance, that periodontal diseases are most frequent, for it is under these conditions that the normal resistance to infection is broken down.

Subnormal function therefore, producing as it does a form of disuse atrophy [5] in the tissues of the periodontium, must be regarded as an important etiologic factor in predisposing these tissues to disease.

FOOD IMPACTION

The impaction of food between the teeth becomes a causative factor in periodontoclasia, inasmuch as it injures the septal gingivæ and robs them of their natural protective function. In this way, lowered resistance to infection occurs, and bacterial invasion, with all that it implies in the production of periodontal disease, is the logical consequence. Under normal conditions food impaction does not occur. In recognition of its possible danger to the tissues of the periodontium, Nature has made elaborate provision to prevent it. This is seen in the interlacing of tooth with tooth by the transeptal fibers of the pericementum. In primitive man, where the conditions of modern life were absent, this was effective in maintaining the teeth in normal proximal contact throughout life. This

was true even in those cases where the diameters of the teeth had been considerably reduced by excessive wear.

The most frequent cause of defective contact points in modern life is proximal caries. Even when these are treated by filling operations, normal contact relations are not always reëstablished and in some instances this is impossible. There are few cases, however, in which they cannot be restored to normal. This should always be done, wherever possible, as it is exceedingly difficult to prevent or successfully treat diseases of the periodontium in which food impaction habitually occurs.

Another cause of abnormal proximal relationships is tooth extraction, which is almost invariably followed by changes in the relative positions of some of the teeth. This is the chief reason why a lost tooth should, as a rule, be replaced. When this is not done and separation occurs, especially in teeth free from caries, it is exceedingly difficult to correct it. The loss of a tooth is, therefore, a much more serious matter than may appear on first thought. In every case it interferes with normal occlusion, and in so doing injures more or less seriously the entire periodontium.

ORAL SEPSIS

Oral sepsis is one of the most important of the local etiologic factors in periodontoclasia, as will be seen when the rôle played by bacteria is clearly understood. (Fig. 26.) Not only does bad mouth hygiene favor the growth and development of bacteria in large numbers, and so contribute to the increased possibilities of infection, it may also induce mechanical and chemical irritation. A further factor in oral sepsis is that of stagnation,[7] which, in the mouth, may be brought about by such conditions as functional inactivity, mal-relation of the teeth and their supporting tissues, the character and rate of flow of the saliva,

etc. The comparative disuse of the teeth, together with the artificial and artificially prepared foods of modern civilization, all tend to the accumulation of débris about and between the teeth, and the production of a condition of stagnation which predisposes to, if in some instances it does not actually cause, disease. The effect of any of these factors, if long continued, is to break down resistance. This applies with especial force when it concerns tissues that are functionally inactive. It may be said, without fear of contradiction, that oral sepsis is always an actual or potential factor in diseases of the periodontium. The importance, therefore, of clean teeth in a clean mouth cannot be overestimated. This is especially true under conditions of modern life, when no one pretends to a normal use of either teeth or jaws, and when, in consequence, a certain amount of degeneration has taken place. The dentist who would prevent the development of periodontal diseases in the mouths of his patients must first learn the practice of oral hygiene. Too often this is confused with the perfunctory cleaning of teeth and relegated to the dental hygienist as though it were a matter of little consequence. There is no one thing in the whole range of dental practice of more importance in the prevention of dental diseases than a clean and well cared-for mouth. Its achievement should be the first concern of the entire dental profession, for by the adoption of a measure as simple as this, and so easily within the ability of all, a very large percentage of the dental ills which now afflict all civilized races could be prevented.

FIG. 26.—Gingival inflammation and hypertrophy in a girl of 16, caused by oral sepsis.

EXCESSIVE OCCLUSAL STRESS (TRAUMATIC OCCLUSION)

"Traumatic occlusion," or, more appropriately, excessive occlusal stress, may be defined as an occlusal force in excess of that which can be borne by the tissues of the periodontium without injury, or as Gottlieb expresses it, "when the tensile strength of the suspensory apparatus is exceeded." The question which naturally arises in this connection is, Why should the perfectly normal act of mastication produce trauma in the periodontal tissues, and by so doing defeat the very purpose for which these tissues were intended? There is no evidence that such ever occurs in the animal world no matter what the wear and tear on the teeth may be. Nor is there any proof that primitive man ever lost his teeth through the development of "traumatic occlusion," notwithstanding the fact that he subjected them to an occlusal stress far in excess of that given to teeth under present-day conditions. What, then, has happened to bring about this change? A partial explanation is, that under conditions of modern civilization a certain amount of degeneration has taken place in the jaws and teeth, bringing about what has been called "disuse atrophy." Neither the jaws nor teeth are as large and robust as they once were. Malformations of the jaws, malocclusion of the teeth, impacted and missing teeth, increased susceptibility to disease, all bear testimony to these degenerative changes, which are so common as to excite almost no comment. The alveolar process, the function of which is to protect the teeth against masticatory strain, is, under present-day conditions, comparatively frail, and in some instances it only partially envelops the roots of individual teeth. When to this is added the increased susceptibility to diseases that tend to break down and destroy these supporting tissues and to thus further weaken them, it is not difficult to understand why the normal act of mastication

may, under these abnormal conditions, cause periodontal trauma. A further explanation is that given by Gottleib [2] to the effect that the eruption of teeth is a more or less continuous phenomenon, which may, under present conditions, bring about abnormal occlusal stress. The belief that when teeth come into occlusion with those of the opposing jaw, the eruptive force has expended itself, he says is incorrect: it may be retarded, but is not at a standstill. "When the teeth have reached the occlusal line, the detachment of the outer enamel epithelium has not yet advanced so far as was formerly believed, namely to the amelocemental junction. In the thirteenth year of age the bottom of the pocket * is generally near the dividing line between the middle and cervical third of the anatomic crown. The last third is still attached to the outer enamel epithelium, and it is very probable that, at this stage, the calcification of these parts may still proceed. After this, the eruption of the tooth advances, but at a slower rate. . . . When the tooth has erupted to the occlusal line, the clinical crown is about two-thirds the length of the anatomic crown. In the course of further eruption, the height of the clinical crown remains, in ideal cases, constant. This is due to the fact that the tooth is worn down on its incisal or occlusal surface to a degree which compensates for the eruption. The length of the clinical crown is the extra-alveolar lever of the chewing apparatus. Conditions being the same, the longer the extra-alveolar lever, the greater the force exerted on the holding apparatus by the act of chewing."

Another source of pathologic disturbance he ascribes to a disturbance in the synchronism of the various elements of eruption: "When, for example, the abrasion of the chewing surface does not keep pace with the downward growth

* By "pocket" Gottleib refers to what in this country is called "gingival crevice."

of the epithelium, the extra-alveolar lever gets longer and longer, and the same force exerted on the tooth puts greater stress on the suspensory apparatus. Further, when all teeth do not erupt at a uniform rate, but one advances faster than its neighbors, this tooth is, as a rule, not worn down correspondingly, and the whole biting strain falls to it." Gottlieb is also of the opinion that the continual deposition of new layers of cementum on the root surface of teeth is an important factor in protecting them against occlusal stress.

It will be obvious to even the most casual that the problem of occlusal stress is a highly complex one, about which the last word has by no means been said. The clinician's attitude toward the whole problem should be one of open-mindedness, meanwhile exercising in practice a position of extreme caution. It is usually the better part of judgment to err on the side of conservatism rather than of radicalism, tinged as it often is by too much enthusiasm.

The conditions which predispose to occlusal trauma (though they may all be present and not cause it) are (*a*) malocclusion; (*b*) faulty operative procedures; (*c*) cuspal abrasion of such a nature as to cause unequal and misdirected pressure; (*d*) size and shape of roots; (*e*) frail alveolar walls; (*f*) interlocking cusps; (*g*) tooth loss; and (*h*) lack of alveolar support due to periodontal disease. It will thus be seen that periodontal trauma may be brought about by two different conditions; one by abnormalities in occlusion, and the other by pathological changes in the tissues that resist occlusal stress. In many instances, probably in most, it will be found that the occlusal force is not so much at fault in the sense of being excessive as are the conditions under which it operates. It frequently happens that a perfectly normal stress is applied to teeth that for some reason or other are incapable of withstanding it with-

out injury. Such conditions may be due to malocclusion, as a result of which the osseous support of the teeth is subnormal through irregular distribution, or, as more often happens, it may be due to the fact that certain pathological changes have occurred in the periodontal tissues themselves, by which they have been partially deprived of their normal resistive function. In other words, the factors which resist occlusal stress have been broken down in the periodontal tissues, through the development in them of disease, rather than that the occlusal force itself is excessive. What had been a perfectly normal stress under conditions of health came to be an abnormal one when the natural support of the teeth had been withdrawn through disease, a condition that may have been brought about without any relation whatever to the occlusion. It requires no stretch of the imagination to understand how, under these conditions, the periodontal tissues may be traumatized, notwithstanding the fact that the stress applied to them in the act of mastication is entirely normal. In many instances there can be no doubt that the phenomenon known as occlusal trauma is due, not to excessive occlusal force, nor to an incoördination of occlusal planes, but to some pathological change in the periodontal tissues themselves, by which they have been robbed of their natural capacity to withstand occlusal stress. The natural reaction to stimulation such as would follow the vigorous use of teeth and jaws is hypertrophy, a condition of supernutrition, by which tissues are, up to a certain point, strengthened to meet the added load which has been placed upon them. Unless this load as applied to the teeth is greatly in excess of normal, or is suddenly thrust upon them as might happen in case of a badly articulated restoration, the tendency is toward increased strength in the periodontal tissues. (Figs. 27, 28, 29.) Regarding this MacMillan says: "When a tooth is under increased occlusal

stress the bone is physiologically adapted to care for that increase. The point where physiological stimulation ends and so-called traumatic occlusion begins, if it ever does begin in the mouth, has not been shown. Before a dental disease produced by a trauma could occur, physiological changes covering a period of years, interpreted radiographically as increased closeness of spacing of trabeculæ, increased density of the compact bone, and alteration in root formation, must have occurred. Paradoxically, in the Roentgen ray negatives used to describe occlusal traumatism, instead of these physiological changes which are associated with increased stimulation, evidence of disuse atrophy, the antithesis of functional stimulation, is more often present. Biologically it is impossible to have physiological changes due to increased function occurring simultaneously with a degenerative change such as disuse atrophy." [5]

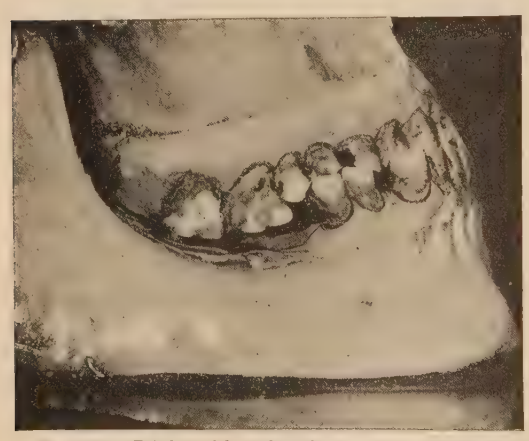


FIG. 27.—Right side of a human jaw, showing excessive occlusal stress on the bicuspids due to loss of lower molars. Age 52. (From Orban, "Tissue Changes in Traumatic Occlusion.")

It is Orban's opinion, based on very careful research and confirmed by similar researches by Gottleib, that "it is not possible that teeth which have been erupted and in good occlusion for from ten to twenty years could suddenly be in traumatic occlusion without any cause. Clinicians sometimes demonstrate traumatic occlusion thus: The patient closes the mouth, the two teeth, for instance two bicuspids,

meet before the others, and the demonstrator claims they are in traumatic occlusion. They say that the trauma is the cause of the loosening. As Gottleib pointed out, this is misleading, for it is quite possible that a few weeks or days previously, the teeth were in normal occlusion. It is not

Fig. 28.—Section through the lower bicuspids shown in Fig. 27. Note the practically normal alveolar process which extends to the cemento-enamel junction. (From Orban.)

possible that two teeth from one to two m.m. longer than the others can exist in occlusion in a jaw for years. The relative increase in length is due to some more or less unknown primary cause. The traumatic occlusion appears as a *secondary factor.* (Italics the author's.) It is evident

FIG. 29.—Apical region of the first bicuspid shown in Figs. 27 and 28: a, dentine; b, cementum; c, alveolar bone; d, normal pericementum. Note "no resorption in bone or cementum, no necrosis, no granulation tissue nor inflammatory change can be seen." (Orban.)

that we must correct the occlusion by grinding down the teeth and must do everything possible to save the supporting structure from the damage caused by the secondary factor—traumatic occlusion." [6] He says further in the same article: "No mention has been made until now of pyorrhea for the reason that pyorrhea means pus flow from the gingival crevice, which has little to do with traumatic occlusion. It may, in a neglected case, leading to secondary damage, be a predisposing factor in the development of a crevice and must always be corrected."

TOOTH LOSS

Tooth loss is another factor to be considered in the breaking down of the periodontal tissues, inasmuch as it interferes with occlusion and tends to the overloading of the remaining teeth. In the case of primitive man, the strain of mastication was distributed over a full complement of teeth, all in normal occlusion. The vigorous use to which they were subjected in mastication served to further strengthen them, just as, conversely, relative lack of use, under modern conditions, adds to their weakness. It is unreasonable to suppose that less than the normal number of teeth will, under these changed conditions, continue to function with the same degree of perfection as that which obtains when they are all present. Especially is this true when it is remembered that the osseous support of the teeth is much less vigorous than that which once prevailed. It is possible to so overload teeth representing less than the normal number, that no matter what their resistive capacity to occlusal stress may be, or how perfect their occlusion, they can be broken down by the strain. And this is what frequently happens when less than the full complement of teeth are required to do the work of the whole, as is so often the case under modern conditions. The force applied

under such conditions, while not of itself excessive, becomes so when applied to less than the normal number of teeth.

There has also to be considered the change in *occlusal relations* which so often follows tooth extraction. The drift of teeth which is more or less inevitable under these conditions, means that the impact from mastication is often received at such an angle as to produce trauma about individual teeth. Then, too, there is the problem of open interproximal spaces to be considered. These are both factors of importance in the etiology of periodontoclasia in addition to that of overloading the teeth. The loss of teeth and their proper replacement is the most serious problem in periodontia, as indeed it is in dentistry, and one which in many instances cannot be solved with entire satisfaction to any one.

Thus it will be seen that there is a type of periodontal trauma that is not due to abnormalities in occlusion, but to abnormalities in the periodontal tissues themselves, as represented by loss of alveolar support and tooth extraction. In other words, the occlusion and the masticating force may both be entirely normal, and yet, because of the weakening of those forces which resist occlusal stress, such as might occur from tooth extraction or periodontal disease, trauma may occur. The trauma produced under these conditions is a symptom, rather than a cause of disease.

The whole subject of "traumatic occlusion" may, therefore, be summed up in the statement that when equilibrium is maintained between occlusal stress and the resistive forces of the periodontium, the act of mastication promotes, rather than destroys, the health of the periodontal tissues; that the most effective way in which to maintain this balance is by the possession of a full complement of teeth in normal occlusion and supported by healthy tissues.

When these conditions prevail, there will be little to fear from cuspal incoördination or excessive occlusal stress.

ABNORMALITIES IN CUSP RELATIONS

In cases of interlocking cusps, uneven occlusal wear, and faulty operative restorations, there may occur sufficient interference with the occlusion to actually produce periodontal trauma. This may also occur in cases of malocclusion. There are few instances, however, in which all the teeth in a given case are so involved, and where operative interference is indicated. A few teeth in a mouth may come into inharmonious occlusal relations through the interlocking of teeth with long cusps, malocclusion, or from occlusal wear, but in those cases in which all the teeth are present and the occlusion normal, this happens infrequently. In many cases in which periodontal trauma is believed to have been caused by some irregularity in the occlusion, it will be found, on more careful study, that the primary factor responsible for the trauma was not cuspal irregularity, but some interference with the forces which resist occlusal stress, such as might occur from tooth loss or alveolar atrophy. Under these conditions the "traumatic occlusion" would be a secondary factor.

RESTORATIVE OPERATIONS

Restorative operations, possibly more than any other one thing under present conditions, are responsible for the production of occlusal trauma. Especially is this true in cases of M. O. D. inlays, crowns and fixed bridges. These are frequently inserted without proper regard for the occlusion, and trauma is produced because of their malrelation to opposing teeth. Trauma may also develop from the fact that there is unequal wear of these restorations from use. This results in leverage or excessive pressure on certain

teeth actually causing a mechanical injury. Great care should be exercised in all operative procedures not to add to the already existing trouble by introducing another, which may cause the loss of both restoration and teeth, as too often happens.

From the foregoing it will be seen that the whole problem of occlusal stress as applied to diseases of the periodontium is a complicated one, and one which is inextricably bound up with both cause and effect. The things to be remembered are, that it is only one of the possible etiologic factors in periodontoclasia and by no means the most important; that as a rule it is neither the occlusion nor the magnitude of the force that is at fault, but a subnormality or weakness on the part of the tissues which resist occlusal stress, and that this has been brought about by disease, tooth loss, malocclusion, etc., and that in those cases in which it occurs it is more often a symptom than a cause of disease.

ETIOLOGIC FACTORS WHICH UNDERLIE AND PLAY AN IMPORTANT PART IN ALL CASES

The etiologic factors which underlie and are more or less common to all cases of periodontoclasia of the inflammatory and suppurative type are two in number, viz.: *bacteria,* plus their toxins and ferments, and the condition of *lowered resistance,* under which they operate. It is Simonton's opinion that "in all cases, the essential etiologic factor is the lowering of the resistance of the soft tissues, permitting the resident micro-organisms to invade. The reaction to this infection will depend on the severity of the local exciting factors; the relative dominance and virulence of the flora of the mouth; the accidental ingress from other mouths of similar species to those existing in the mouth of the case being considered, etc. The artificial care of the

mouth and the function will also be factors."[8] Suppurative periodontoclasia or so-called "pyorrhea alveolaris" could not exist without the concurrent influence of bacteria and low resistance. Except in the atrophic type already described and believed to have its primary cause in some nutritional disorder, bacteria and the associated condition of lowered resistance are the two most important factors in the etiology of periodontoclasia. Lowered resistance and bacterial infection characterize all cases. Control these, and practically all could be prevented. In treatment, operative and postoperative, their control is of first importance, without which no other treatment will avail much. Regarding this Hartzell says: [3] "That type of periodontoclasia characterized by pyorrhea can be prevented and cured in every instance in which we can control the growth and invasion of these pyogenic organisms." In enumerating the etiologic factors in periodontoclasia, Hartzell includes all those "that tend to break down the normal resistance of the tissues to the invasion of pyogenic bacteria. These are legion, and because of the fact that superficial investigators in clinical lines have noticed that correction of malocclusion or dietetic change has resulted in the cure of a case of pyorrhea, they have confidently stated that the cause of the condition is this or that particular factor which they have noticed and have had the fortune to eliminate, not recognizing the fact that they have merely increased tissue resistance by removing a handicap. I have conclusively shown that pyorrhea cannot exist without pyogenic bacteria; that individuals may be on the worst possible diet, and, notwithstanding, if the necks of the teeth adjacent to the soft tissues are kept sufficiently free from bacteria, and the soft tissues are daily stimulated by friction and pressure, no periodontoclasia, or pyorrhea, can exist." [3]

The pyogenic organisms referred to by Hartzell [3] as being the chief agents in the etiology of periodontoclasia are the streptococcus and staphylococcus, the more important of which is the streptococcus. They are possessed of powerful invasive properties, making possible their penetration into any tissue in which resistance to bacterial infection is subnormal. Simonton says, "The evidence of research on the bacteriology of parodontoclasia supports the view that the inflammation is secondary to a lowering of resistance of the soft tissues which permits resident organisms to invade. The factors which produce loss of tone are in all probability endogenous. Changes of saliva, which may be brought about by the same or other endogenous factors, would doubtless affect the balance of the micro-organic flora and vary relative dominance and virulence." [8] While the streptococcus and staphylococcus are the chief etiologic factors in suppurative periodontoclasia, they are not causative in the sense that the typhoid bacillus is causative of typhoid fever. Their part in the etiology of periodontoclasia is due in large measure to low cell resistance of the periodontal tissues and to their peculiar invasive property. But for these, they would remain as harmless residents of the mouth. Because they are not specific, bacterial vaccines are of no value in treatment.[4] It is this lack of specificity which makes their use illogical.

A *subnormal resistance* to infection on the part of the cells composing the tissues of the periodontium is a natural corollary to functional inactivity. The failure to vigorously use the teeth and jaws in mastication makes for poor circulation and undernourishment. Such conditions may be purely local and be the result of local conditions, as is often the case with the tissues of the periodontium. This being true, it logically follows that, in the prevention and treatment of periodontal disease, it is of first importance

that the circulation of the periodontal tissues be increased through stimulation, to the end that they be better nourished and that their resistance to infection be raised. This is of more importance, even, than the control of the bacterial flora of the mouth, for if resistance to infection be maintained at a high level, no matter how abundant the bacteria, they will not cause infection. All efforts, however, at prevention and treatment should be directed toward both the reduction of bacteria and the building up of those forces which resist infection.

REFERENCES

[1] Fleischmann, L., and Gottleib, B., 1921, "A Contribution to the Histology and Pathogenesis of Pyorrhea Alveolaris," *Dental Cosmos,* vol. 63, p. 386.

[2] Gottleib, B., 1928, "The Formation of the Pocket: Diffuse Atrophy of Alveolar Bone," *Journal American Dental Association,* vol. 15, pp. 463, 464, 466.

[3] Hartzell, Thos. B., 1927, "Important Factors in the Etiology and Control of Periodontoclasia," *Journal American Dental Association,* vol. 14, pp. 899, 901.

[4] Hirschfeld, I., 1926, "An Investigation of Inava. Endocorps Vaccines," *Journal American Dental Association,* vol. 13, p. 1613.

[5] MacMillan, H. W., 1925, "The Clinical Application of Some Anatomical Facts to Dental and Oral Surgery," Reprint, p. 6.

[6] Orban, B., 1928, "Tissue Changes in Traumatic Occlusion," *Journal American Dental Association,* vol. 15, pp. 2101, 2104.

[7] Simonton, F. V., 1927, "Parodontoclasia," *The Pacific Dental Gazette,* April, 1927.

[8] Simonton, F. V., 1927, "The Etiology of Parodontoclasia," *Journal American Dental Association,* vol. 14, pp. 1790, 1791, 1810, 1827.

[9] Smith, A. Hopewell, 1911, "Pyorrhea Alveolaris—Its Interpretation," *Dental Cosmos,* vol. 53, p. 410.

[10] Symposium, 1928, "Etiologic Factors in Periodontal Lesions," *Journal American Dental Association,* vol. 15, p. 504.

CHAPTER VIII

TREATMENT OF PERIODONTOCLASIA

Of first importance in the consideration of the subject of periodontoclasia is its treatment. It is the only thing of interest to the patient, who is concerned not with its etiology or pathology, but whether, as a patient, he can be cured. To a considerable extent this is true of the practitioner, whose interest in the course and progress of disease is primarily that of the clinician who studies its various manifestations and causes, in order that he may the more successfully treat it. All the investigations carried on by the research worker in his laboratory; all the clinical observations made by the practitioner in his daily contacts with his patients, are of value chiefly as they make the successful treatment of disease more certain. For these reasons the subject of treatment is of great interest and in the case of periodontoclasia takes on an added importance since it is a subject about which there has long been considerable confusion.

EXAMINATION

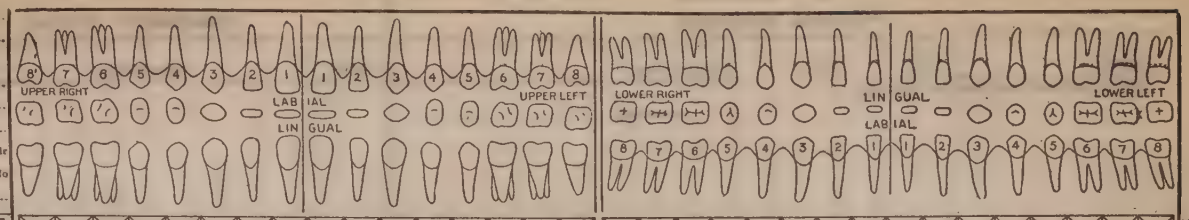
As a preliminary to treatment, a complete radiographic examination should be made of all cases, for in no other way can a working knowledge be gained of the extent of bone loss about the involved teeth. Such radiographic examination will not, of course, tell the whole story, since bone loss limited to the lingual surface of a root may not be represented in the radiogram. Nevertheless, it is of

great assistance and should always be made a part of the examination, inasmuch as it affords a fairly accurate knowledge of existing conditions. By it one determines the extent of alveolar atrophy as it involves the buccolabial surfaces and interproximal spaces; the condition of the periapical tissues; the presence or absence of root ends, pulpless teeth, unerupted teeth, etc. Referring to the radiographic examination of a case of periodontoclasia in its early stages in which there were "absolutely no clinical signs" of disease, Pollia says: "I wish to take this opportunity to state that radiographic examination will reveal more of interest to the oral diagnostician in cases of this character than the oral examination. It is not unusual for me to hear from patients, who have just been given a grave prognosis in regards to the possibility of saving certain teeth which have been the victims of a slow and insidious destruction of the alveolar process, that they cannot imagine such a thing to be true, since they have visited the dentist every six months for years, and have been pronounced, dentally, in good health, and have always taken the best of care of their teeth. That is undoubtedly true, but they have been instructed only as far as their dentists knew. Any dentist who professes to practice preventive dentistry, and limits his or her investigation to an inspection of the teeth every six months and the repair of local damage, or scaling of the deposits, without a complete radiographic examination of all the teeth, is not protecting the oral health of those patients to the fullest extent, because, when a great many of these conditions reach the point where they excite local signs, the diseased areas are probably beyond the therapeutic skill of the average person in general practice. Too many radiographs like this one (showing the beginnings of alveolar absorption) pass as normal. I see many teeth which have just been to the periodontist which look no

better than these where the calculus has eluded the delicate touch of the skilled fingers, and where no attempt is made to correct the underlying influence. This is not to be taken as an indication of inefficiency on the part of these specialists, but as a sign that eyes, fingers and experience have their limitations, and that these limitations can be extended by the radiograph, and that there can be no reason for not giving many of the patients the benefit of it. . . . This degree of perialveolar and periodontal pathological abnormality is of utmost importance to the periodontist, and should demand his closest attention because he has means at his disposal which can arrest or retard this type of disease. The location of early caries is important, but unless something is developed in the near future which will arrest caries when first demonstrated radiographically, it can only serve as a guide for future operative efforts, because a certain amount of tooth structure must be cut away before a satisfactory restoration can be made. The periodontist, however, is in a better position to take advantage of these radiographic hints, because by instrumentation, brushing and careful adjustment, good results can be made to follow immediately." [30] With these observations by Pollia the author is in entire agreement, for they confirm in every particular his almost daily experiences. He cannot be too emphatic in urging the importance of radiography in the diagnosis and treatment of periodontoclasia. But of more importance even than this is the regular and systematic radiographing of the teeth and periodontal tissues of all patients for the purpose of *preventing* disease. This cannot be accomplished with entire satisfaction to anyone when dependence is placed upon clinical examinations, no matter how thorough these may be; the early changes taking place in the alveolar process are far too subtle to be detected by the unaided eye. And there can be no excuse for neglecting

Case No..... M..... Address..... .....19.....

X-Ray No. .....
Phone.....
Referred by
.....
Age..... Sex.....
Occlusion Class.....
Clean—Yes—No—Fair
Use of Brush Yes—No
1-2-3 Times Daily....



Dental Disease — Labial / Distal / Lingual
Vitality Test
Traumatic Occlusion
Defective Contact
Marginal Injury
Mobility
Diagnosis — Periapical / Parietal
Prognosis — Periapical / Parietal
Treatment Recommended

DENTAL DISEASE
1 Erosion
2 Abrasion
3 Etched enamel
4 Hypercementosis
5 Root absorption
6 Impaction
7 Non-Eruption
8 Hypersensitiveness
9 Exposed pulp
10 Calcific degeneration

PERIODONTAL ETIOLOGY

TO—Traumatic Occlusion
1 Malposition
2 Defective restoration
3 Abnormal tooth form
4 Defective contact

MI—Marginal Injury
1 Deposit
2 Defective restoration
3 Abnormal tooth form
4 Defective contact
5 Incorrect mouth toilet
6 Operative trauma
7 Anatomical defect
8 Inurious habits
9 Decomposing debris
10 Chemical injury

PERIODONTAL DIAGNOSIS

Periapical condition
1 Radio-normality
2 Circumscribed radiolucency
3 Diffuse radiolucency
4 Circumscribed radioparency
5 Radiopacity

IB—Infected bone
IR—Imbedded root

PA—Parietal Abcess
1 Pericemental
2 Gingival

R—Recession
1 Traumatic
2 Atonic

G—Gingivitis
1 Simple chronic
2 Hypertrophic
3 Subacute
4 Phagedenic

P—Pericementoclasia
1 Inflammatory
2 Sub-Inflammatory

A—Alveoloclasia

M—Mobility
1 Slight
2 Moderate
3 Marked

RECOMMENDATION
Ex —Extraction
Cur —Curretage
Dv —Devitalization
Rt —Root canal treatment
APC —Apicoectomy
PT —Periodontic treatment
Cr —Crown
FL —Filling
FXB —Fixed bridge
RMB—Removable bridge
Den —Denture

KEY

Record recession by a line—pockets by a shaded area on chart.

Record caries by outlining on tooth or on diagrammatic chart opposite "dental disease". Record other dental diseases by key number in same column.

Record vitality, also prognosis as +, —, or ?

Record traumatic occlusion, defective contact, marginal injury, mobility and periapical condition by check mark (√), and parietal condition by initial. Key number may be added if greater detail is desired.

When extracted draw line through tooth. Mark crown—C, bridge abutment—B.

Local Tonicity.....Bacterial Finding.....

Systemic Disease [if exhibiting oral manifestations].....

Periodontium—Normal.....Ischemic.....Inflamed .....Suppurating.....

Dentifrice Prescribed.....Mouth Wash Prescribed.....Instructions.....

REMARKS

FIG. 30.—Examination and record chart prepared by the American Academy of Periodontology.

SUBSEQUENT EXAMINATION

DATE	RECURRENCE OF: SOFT DEPOSIT	HARD DEPOSIT	STAIN	PERIODONTIUM: HYPEREMIA	ISCHEMIA	PUS	SALIVA: THICK	THIN	NORMAL	DIET: CARBOHYD.	PROTEIN	VITALITY TEST AND RECURRENCE OF CARIES — INDICATE VITALITY AS + OR —; CAVITIES BY SURFACE NUMBER OR CHECK MARK. 1-OCCLUSAL 2-MESIAL 3-DISTAL 4-LABIAL OR BUCCAL 5-LINGUAL: UPPER RIGHT 8 7 6 5 4 3 2 1	UPPER LEFT 1 2 3 4 5 6 7 8	LOWER RIGHT 8 7 6 5 4 3 2 1	LOWER LEFT 1 2 3 4 5 6 7 8

PHYSICAL DIAGNOSIS

DATE | AGE | SEX | OCCUPATION

SYMPTOMS AND NATURE OF DISEASE

PHYSICIAN. ... .

HISTORY - FAMILY AND PERSONAL.

PHYSICAL EXAMINATION:

TONSILS

NASAL SINUSES

PELVIC ORGANS

GASTRO-ENTERON

KIDNEYS

HEART

BLOOD PRESSURE
DIAST. SYST.

HEMOGLOBIN

BLOOD COUNT

BLOOD CHEMISTRY

REMARKS:

TREATMENT RECORD

DATE	TREATMENT	RESULT

ESTIMATE RECORD

	TOOTH	RECOM.	DENTAL OPERATIONS	PROSTHETIC OPERATIONS	EXAMINATION
UPPER RIGHT	8				RADIOGRAPHS
	7				EXAMINATION AND CASE RECORD
	6				STUDY MODELS
	5				REMOVE CR. AND BR.
	4				DENTAL HYGIENIST
	3				MATERIALS
	2				LABORATORY
	1				
UPPER LEFT	1				PERIODONTIA
	2				
	3				
	4				
	5				
	6				
	7				
	8				
LOWER RIGHT	8				ORTHODONTIA
	7				
	6				
	5				
	4				
	3				
	2				
	1				
LOWER LEFT	1				ORAL SURGERY
	2				
	3				
	4				
	5				
	6				
	7				
	8				

ARRANGEMENT FOR FUTURE APPOINTMENTS:

EXAMINATION, ETC.	
DENTAL OPERATIONS	
PROSTHETIC OPERATIONS	
PERIODONTIA	
ORTHODONTIA	
ORAL SURGERY	
ESTIMATE TOTAL	

LEDGER RECORD

DATE	TOOTH	OPERATION	DEBIT	CREDIT

to do so since the cost of radiographic examinations is in no case prohibitive.

HISTORY CHARTS AND STUDY MODELS

Following the radiographic examination, a vitality test should be made of all doubtful teeth to ascertain their status. An inquiry regarding the patient's general health should be made with a view to its possible relation to the conditions seen in the mouth. This should be followed by a most minute and painstaking examination of the entire mouth and adjacent tissues, the data so obtained to be carefully recorded on a chart provided for that purpose, and made the basis for subsequent treatment. Many charts have been arranged for the recording of such data and are available to anyone wishing to make use of them.[1] If not entirely satisfactory to meet one's requirements, they can be used as a guide to such changes as may make them more acceptable. It will be observed from the accompanying illustrations that provision has been made to meet every contingency that may arise. (Figs. 30, 31, 32.) To some they may seem unnecessarily detailed, especially to the general practitioner whose interests are chiefly those of the clinician. Wherever it can be done, however, it is desirable to make them as complete as possible, as they provide a storehouse of very valuable and much-needed information. In this respect, the clinician in this field has lagged behind the research worker. A very comprehensive plan for mouth examination is outlined by Simonton.[32] All hopelessly involved teeth, including pulpless teeth with periapical infection, should be extracted before treatment for periodontoclasia is begun. In those cases in which the standard of hygiene is low, a prophylactic treatment should precede operative treatment. Indeed, it is often desirable that this be done before beginning the examination or the

taking of impressions for study models. In those cases in which the occlusion has been seriously interfered with either through extraction or malposition, study models should be made. Not only are these helpful in the diagnosis and treatment of all cases, but when carefully made, they furnish a valuable future record of the case which can be obtained in no other way. Also when a large collection is secured in this manner, they provide scientific data of no little value. Good plaster models, however, are not easily obtained in all cases, and, when carefully made from plaster impressions, sometimes involve the patient in a disagreeable experience and added expense which in cases not absolutely essential to diagnosis and treatment it may be the part of wisdom to forego. Models made from hurriedly taken wax impressions may serve all practical purposes, but because they lack certain details they possess little of scientific value. A wise attitude toward the subject is to secure as many good models as one can, and insist upon their being made in those cases essential to correct treatment and in those presenting features of more than usual interest. When the general health is involved, the coöperation of the patient's family physician should be enlisted. It is never wise to undertake the medical treatment of any patient. This should always be left to the physician, who, because of his training, is better qualified than anyone else to undertake such treatment. The best results are obtained in these cases through the coöperation of both dentist and physician.

THE SCOPE OF TREATMENT

Treatment has for its purpose two objectives: *First,* the correction, by operative procedures, of the pathologic conditions already existing in the periodontium; and *second,* the elimination through operative and postoperative care of

CASE NO. M

ORAL DIAGNOSIS CHART

19

COMPLAINT

GENERAL CONDITION OF MOUTH

		CLINICAL FINDINGS								RADIOGRAPHIC FINDINGS									TREATMENT & OPERATIONS INDICATED		
													ALVEOLAR PROCESS ATROPHY								
		GINGIVA	CARIES	CARIES X-RAY	RESTORATIONS	TRAUMATIC OCCLUSION	MOBILITY	VITALITY	CONTACT POINT	PULP CHAMBER AND CANALS	PERICEMENTAL MEMBRANE	PERICEM. LAMELLA	CREST	BIFURC.	APICAL CIRCUM.	APICAL DIFFUSE	HYPERTRO-PHY		STOMATOLOGY		DENTAL RESTORATIONS	
UPPER RIGHT	8																	8				
	7																	7				
	6																	6				
	5																	5				
	4																	4				
	3																	3				
	2																	2				
	1																	1				
UPPER LEFT	1																	1				
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	4																	4				
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	1																	1				
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	4																	4				
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	6																	6				
	7																	7				
	8																	8				

PERIODONTIA

EXAMINATION STOMATOLOGY RESTORATIVE TOTAL

FIG. 31.—Oral diagnosis chart as prepared by Dr. Elmer S. Best.

Pages 2 and 3

CASE RECORD

Age Weight Referred by

Physician

Date	Reported by	History	Findings	Diagnosis	Treatment
		DENTAL HISTORY			
		DENTAL CARIES			
		EXTRACTION OF TEETH			
		TEETH DEVITALIZED			
		PERICLASIA			
		PERICLASIA TREATMENT			
		ORTHODONTIC TREATMENT			
		THROAT, NOSE, EAR AND SYSTEMIC HISTORY			
		GLANDS OF NECK			
		TONSILITIS ACUTE			
		CHRONIC			
		PHARYNGITIS			
		LARYNGITIS			
		NASAL OBSTRUCTION RIGHT		LEFT	
		NASAL DISCHARGE ANT. RIGHT		LEFT	
		POST NASAL DISCHARGE			
		EAR CONDITIONS—IMPAIRED HEARING		TINNITUS	
		DISCHARGE		PAIN	
		EYES—VISION		INFLAMMATION	
		HEADACHE			
		NEURITIS FACIAL			
		NEURITIS GENERAL			
		JOINT CONDITIONS			
		MUSCLE CONDITIONS			
		GASTRO INTESTINAL—APPETITE		MASTICATION	
		DIGESTION			
		HEART			
		BLOOD		EXCESSIVE BLEEDING	
		RESPIRATORY			
		KIDNEY			
		NERVOUS			
		DUCTLESS GLANDS			
		GENERAL HEALTH		ENERGY	
		FATIGUE		SLEEP	
		SUMMARY			
		REPORT ON NOSE, THROAT, EAR AND SYSTEMIC EXAMINATIONS			
		GLANDS OF NECK			
		TONSILS			
		PHARYNX			
		NASO-PHARYNX			
		LARYNX			
		NOSE RIGHT			
		LEFT			
		EARS RIGHT			
		LEFT			
		TRANSILLUMINATION OF SINUSES			
		X RAY OF SINUSES			

ORAL PROPHYLAXIS CHART

DATE	PRESENCE OF					NEW CAVITIES						TOOTH BRUSHING									DIET								
	FOOD	CALCULUS	STAIN	SUPPURATION	EROSION	OCCLUSAL	PROXIMAL	LABIAL BUCCAL	LINGUAL	SALIVA	FREQUENCY	OCCLUSAL	PROXIMAL	LABIAL BUCCAL	LINGUAL	DENTAL FLOSS	DENTIFRICE	MOUTH WASH	SUGAR	STARCH	PROTEIN	FAT	FRUIT	BEVERAGES	HABITS ACTIVE SEDENTARY	TOBACCO
EXAMINATION																												
1																												
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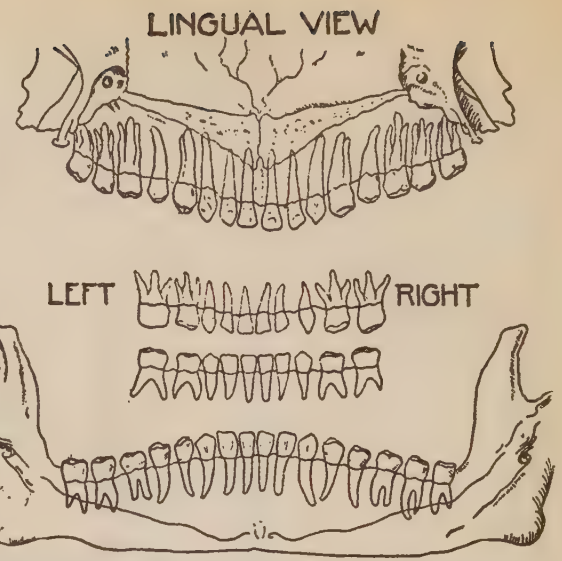

INSTRUCTION IN HOME CARE OF MOUTH

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13
14
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16
17
18

the underlying causes of disease, to the end that health of the periodontal tissues be reëstablished and maintained. This dual nature of treatment should not be overlooked, for if it be limited to either, a full measure of success is impossible. This involves the doing of two things both of which require the earnest and persistent coöperation of the patient: (*a*), the establishment and maintenance of a high standard of mouth hygiene, by which the bacteria that play such an important rôle in the etiology of periodontoclasia are reduced to a minimum: and (*b*), the regular and persistent stimulation of the periodontal tissues with a view to increasing their resistance to bacterial infection. These are of such importance in the prevention, treatment and postoperative care of all cases of periodontoclasia that they cannot be over-emphasized. They are the means by which a cured case is maintained in health, for if the mouth is allowed to become unhygienic and the periodontal tissues are not given the stimulation so essential to their health and well-being, reinfection is almost inevitable, no matter what else may be done. The unfortunate feature in this aspect of treatment is that one is so dependent for success upon the intelligent and persevering coöperation of the patient. It is essential, therefore, that he be made to understand this when he applies for treatment, and that he be carefully instructed in the most exact methods of oral hygiene. This will be considered in detail later.

SUBGINGIVAL CURETTAGE

Operative treatment consists in the subgingival curettage of the involved root surface to the depth of each pocket, removing from it all calculary deposits, and, what is equally important but more often overlooked, the septic material that is always present on the surface of the root.[34] This, in reality, is the *sine qua non* in treatment, and the point

at which most failures occur. It requires a definite and painstaking technique and the use of instruments especially designed for that purpose. Both will be described later. The belief held by many that the only requisite to success in this operation is the removal of subgingival tartar, and that this can be done with any set of scalers, is a serious mistake and one which is bound to end in failure. Another error commonly held and one which has been fostered by the nostrum makers, is that certain medicinal preparations are important in the treatment of these cases. "Just scale the teeth," so the directions read, and apply this or that particular remedy, and, *ipso facto,* the thing is done. There is no greater fallacy. It is unfortunate, perhaps, that it cannot be done in this "happy-go-lucky" way, but long experience by many operators has proved beyond a doubt that it cannot; there is no easy road to success in periodontia.

All of the root surface to the depth of each individual pocket must be gone over with instruments especially designed for that purpose. Nothing can be taken for granted. This is not so much a scaling operation as a delicate planing of the root surface, designed to superficially remove its more or less septic and roughened surface. Care should be exercised not to cut deeply, for in so doing the dentine will be exposed and sensitiveness may develop. In spite of care, this undoubtedly happens in some cases without apparently interfering seriously with results.

It is desirable to follow some definite plan in treatment. Begin, for example, with the last tooth on the lower right side, curetting the distal surface of the involved root and finishing this surface before proceeding to another on the same tooth.[8] The treatment of each tooth should be finished before passing to the next, except in those cases where

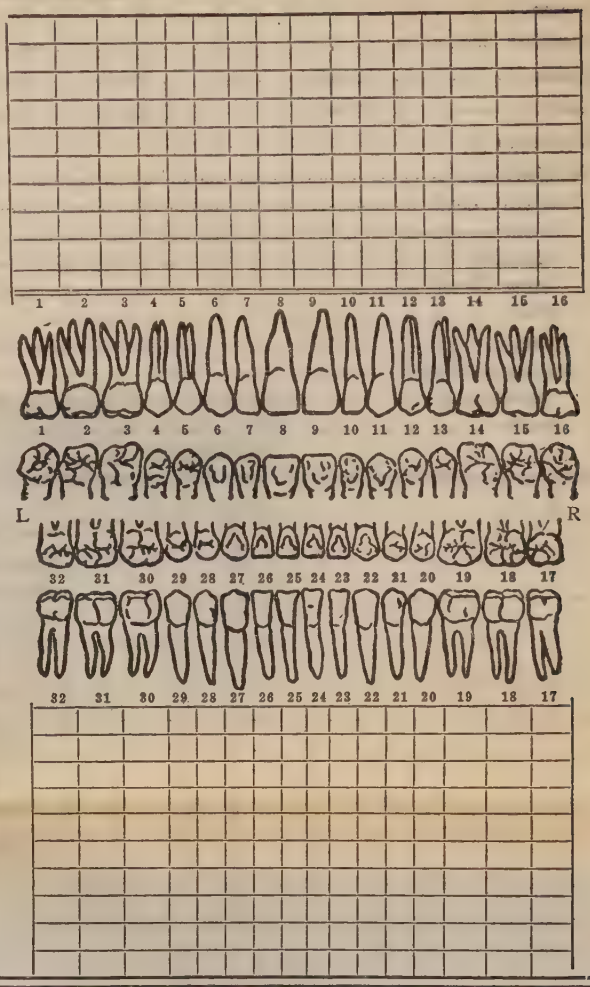
No..... Date.....

Name..... Address.....

Referred by..... Res. Phone..... Bus. Phone.....

1.....Observe .....
2.....Etched Enamel .....
3.....Hypersensitive .....
4.....Erosion .....
5.....Defective Contact .....
6.....Excess Occlusal Area.....
7.....Occlusal Stress .....
8.....Resorption ..... 1—2—3—4 .....
9.....Vitality ..... + or — .....
10.....Mobility ..... 1—2—3—4 .....

a.....Radiograms .....
b.....Study Casts .....
c.....Radiographic Checkup .....
d.....Deposits ..... 1—2—3—4 .....
e.....Gingivitis ..... 1—2—3—4 .....
f.....Vincent's Infection ..... 1—2—3—4 .....
g.....Periodontoclasia ..... 1—2—3—4 .....
h.....Instruction in Mouth Care.....
i.....Mouth Brush .....
j.....Mouth Wash .....
k.....Dentifrice .....
l.....Tongue Cleanser .....
m.....Suggested Visits .....



10.....Mobility ..... 1—2—3—4 .....
9.....Vitality ..... + or — .....
8.....Resorption ..... 1—2—3—4 .....
7.....Occlusal Stress .....
6.....Excess Occlusal Area.....
5.....Defective Contact .....
4.....Erosion .....
3.....Hypersensitive .....
2.....Etched Enamel .....
1.....Observe .....

FIG. 32.—Examination and record chart by Dr. Donald F. Mosher. As described by him "the chart of the teeth on reverse side is for recording everything found in patient's mouth at first visit, including missing teeth, restorative work, cavities, pockets, and all information of interest. The front page is to record subsequent cavities, pockets or other conditions that may arise, giving one a continuous check on the mouth by comparison."

Age .....
Weight .....
Complaint .....
Health..... Good..... Poor.....
Habits..... Active..... Sedentary.....
Elimination .....
Nutrition .....
Protective Foods .....
Fruits .....
Vegetables .....
Coarse Cereals .....
Milk .....
Water .....
Imbalanced Foods .....
Carbohydrates .....
Proteins .....

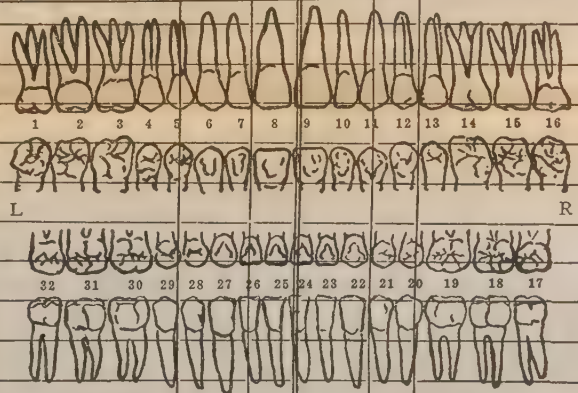
Breathing..... Normal..... Mouth.....
Mastication .....
Lips..... Normal..... Pale.....
Tongue..... Normal..... Coated.....
Saliva .....
Care of Mouth.....
Blood Pressure .....
Blood Examination:
R. B. C.....000; W. B. C.....; Hb. .....%
Differential Count:
P M. N.....%; S. M.....%
P. M. E.....%; L. M.....%
P. M. B.....%; Trans.....%
Smear .....
Urinalysis:
Sp. Gr.....1.0.....; Albumen.....; Sugar.....

Mic. Examination .....

Occupation _____ Employed by _____

Physician _____ Referred to _____

A. Amalgam	B. Bridge		Ca. Carie	D. Denture
Ab. Abrasion	C. Crown		Ce. Cement	E. Extract
Er. Erosion	G.P. G. Percha		P. Porcelain	S. Silicate
G. Gold	I. Impacted		R. Residual	X. Missing



Date of Removal
Abscessed A

Transillumination
1+ to 4+

Pulpless—P
Pyorrhea—PY

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17

REMARKS:

the pockets are not so deep but that several teeth may be completed in a single treatment. In such cases the same surface of several teeth may be curetted with the same instrument before taking up the next; the same plan may be repeated by curetting another surface of several teeth with the second instrument before passing to the third, and so on until all the teeth under treatment be completed. This should be done in such a way as to make further operative treatment at subsequent visits unnecessary. This is chiefly in the interest of efficiency, for treatment may be repeated and should be, in those cases in which the first treatment has failed, as sometimes happens. There can be no doubt, however, that the completion of all operative procedures at a single treatment is desirable, since the repeated curretage of pockets interferes with the healing process. Moreover, it tends to increase the sensitiveness of root surfaces. The more experienced and skillful the operator, the less often will he find it necessary to repeat treatment at subsequent visits. Having begun at one definite point, treatment should be continued until each tooth in that jaw, passing from one side to the other, has been treated. When all the teeth of one jaw have been completed, treatment of those in the other should be begun and carried forward until the entire mouth has been given

FIG. 33.—Bone repair (lower) following treatment of deep periodontal pocket complicated by a periodontal abscess. Note healthy appearance of the bone. Treated by T. Sidney Smith.

the necessary treatment. This phase of treatment is given in detail in the chapter on Instrumentation.

It is necessary that the hand of the operator be firmly fixed in order to control the proper use of the instrument. This is of great importance, since failure to do so explains much of the pain attending treatment. The movement of the instrument should always be under the control of the fingers, never the wrist or arm. In much of the operative treatment done on the mandible, the field of operation is exposed by the left hand, upon which the right hand firmly rests while engaged in the curettage of pockets. Each one must work out for himself his own technique, but in doing so should remember that the right hand must always be firmly fixed before beginning operation if he is to inflict the minimum pain upon his patient at the same time that he achieves the maximum of results. It is also important that a pull, rather than a push stroke, be employed in curetting the root surface. The patient instinctively recoils from a push stroke even when the instrument is under perfect control. There is always present the subconscious fear that the instrument is going to slip beyond the confines of the pocket and so cause pain. Except in rare instances, the push stroke is never necessary. As a rule the curettage of the alveolar process at the bottom of the pocket is not necessary. Except for the ragged edge

Fig. 34.—Upper: deep pocket on distal surface of left cuspid; presented with chronic periodontal abscess discharging through fistulous opening. Lower shows bone repair several years later. (From T. Sidney Smith.)

which will be absorbed as a part of the healing process, it is important that as much of the bone as is possible be retained. Just as the balance of the alveolar process will be absorbed without an alveolectomy following the extraction of teeth involved in periodontoclasia, so will the alveolar border take care of itself by absorption following curettage of the periodontal pocket.

The introduction into the pocket of some drug or preparation for the purposes of stimulation, or the destruction of the epithelial cells which may line the inner wall of the soft tissues, has often been tried, but always with uncertain results. As a rule they probably do more harm than good. Generally speaking, all that is necessary in the way of tissue stimulation and the breaking up of the epithelial lining will be done by instrumentation more satisfactorily than by the use of any drug or combination of drugs at present known. Drugs play a very limited part in the treatment of periodontoclasia.

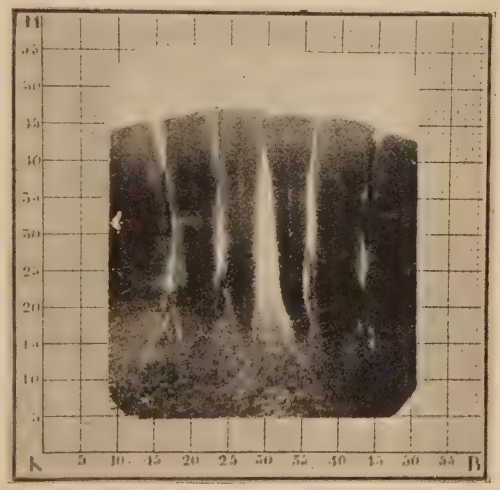


FIG. 35.—Resorption of alveolar bone due to advanced periodontoclasia. (From "Studies in Periodontal Pathology," by H. K. Box.)

Regarding this Kirkland says: [39] "Another cause for failure in the treatment of these conditions is the reliance on drugs to accomplish the results desired. Medicinal remedies are ineffective in this disease (periodontoclasia) and should be given no consideration whatever. We may use

mild antiseptics to irrigate affected tissues as a general routine in surgical treatment, but strong drugs are not only unnecessary, but are also positively contraindicated, and the operator who persists in their use will defeat his own efforts. There has always existed a false impression among practitioners of dentistry, as well as in the mind of the laity, that disease of the periodontium can be cured by the use of mouth washes or the application of strong drugs to the diseased areas. The sooner we get the drug-treatment idea out of our minds, the sooner we will be in position to render worthwhile service to those who entrust the health of their mouths to our skill and judgment."

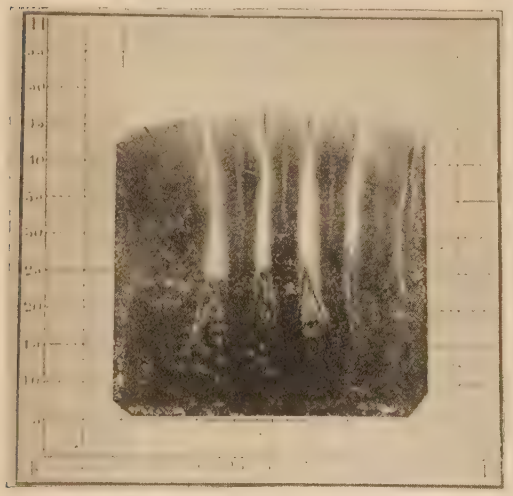


Fig. 36.—Showing formation of new bone one year after treatment. Note clearly defined lamina dura. (From "Studies in Periodontal Pathology," by H. K. Box.)

When the treatment as outlined above (known as the conservative method) is skillfully carried out, the results are most satisfactory, though they are not to be compared in value to prevention. As a rule the bone loss is irreparable, though occasionally there will be more or less regeneration, and in many cases an increase in density in that which remains. (Figs. 33, 34, 35, 36.) There is usually some recession of the gingivæ with exposure of the root surface. In certain cases there will be obliteration of the pockets by reattachment of the periodontal tissues to the root surfaces

(Figs. 37, 38.) In cases successfully treated, infection ceases and health is reëstablished. In those not too far advanced, this can be accomplished without mutilation of the gingivæ or the use of an anæsthetic. In cases of nervous or highly sensitive patients, there is no reason why a local anæsthetic may not be used, and in cases of deep pockets this may be desirable. Carefully done, the operation of subgingival curettage is not painful, and where it can be successfully practiced, exceeds in value any method of treatment at present known.

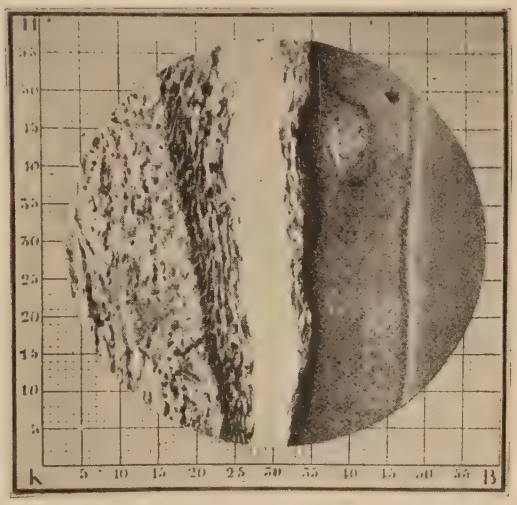


FIG. 37.—Showing reattachment of the connective tissues following subgingival curettage. Note the reattachment of the epithelium to the cementum. (From "Studies in Periodontal Pathology," by H. K. Box.)

CORRECTION OF OCCLUSION

In considering the treatment of occlusal stress in its relation to periodontoclasia one should always bear in mind "that we are concerned with a biologic problem and that the imperfection in occlusal relations or alignment of the teeth that carries a case just beyond the limits of type may not indicate a perversion of physiological processes." [21] In his article on "traumatic occlusion," Hatfield calls attention to the fact that "in occlusion we are concerned with parts of a living organism, and as such they exhibit the natural variations and defects that characterize all struc-

tures."[16] In considering, therefore, the treatment of occlusal stress one should not overlook the fact that there is no such thing as a normal occlusion in the sense so often conceived of by both periodontists and orthodontists.[18] An "ideal occlusion is a creation of the imagination."[16] For these reasons, what appears to be an abnormality in a given case may in reality be entirely normal. If in the treatment of "traumatic occlusion" this be kept clearly in mind, there will be less danger from overenthusiasm regarding its correction. As already pointed out, periodontal trauma may occur under two conditions: one, in which the primary factor is some abnormality in occlusal relations such as might happen in cases of faulty restorations, interlocking cusps, uneven occlusal wear, tooth loss, etc.; the other, cases in which the periodontal tissues have been so weakened by disease as to be incapable of withstanding a normal occlusal force. In the treatment of those cases falling within the *first* group, the correction of occlusal defect is obviously the first duty. When produced by faulty restorations of any kind, these should be made to conform to correct occlusion by either reconstructing them altogether, or grinding their occlusal

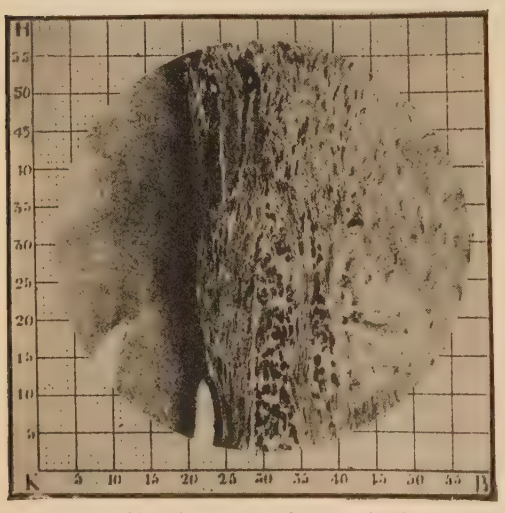


FIG. 38.—Showing reattachment farther down in same pocket. (From "Studies in Periodontal Pathology," by H. K. Box.)

surfaces. These are examples of cases that may produce trauma of so pronounced a type as to break down rapidly the involved teeth before they have had time to adapt themselves to the suddenly increased load which has in this way been thrust upon them. Fixed bridges, the attachments of which are not in line with their anatomical axes, may so change the positions of the supporting teeth as to cause serious trauma without direct reference to the impact of occlusion. In cases of interlocking cusps, uneven occlusal wear, etc., freedom of motion should be provided by careful tooth-grinding. To do this, first test the teeth in centric occlusion with a good carbon paper. Then, with a small carborundum stone, grind lightly the exact points of contact, including those of the opposing teeth. Next test them in the different excursive motions of mastication and again grind very lightly. This does not mean that they should be ground to conform to any possible relationship which they may assume by throwing the mandible outside of its normal masticatory limits as is sometimes done. Except in exaggerated cases, the tooth should never be entirely disoccluded; there are few cases that require radical grinding, and in the group under consideration, almost none.

In those cases of occlusal stress produced by tooth loss, the treatment is primarily that of reducing the load on the remaining teeth by the use of artificial substitutes. In many instances, possibly in all, this cannot be done with entire satisfaction, since, under the force of occlusion, the artificial teeth yield sufficiently to throw the greater part of the load upon the natural teeth. It remains, however, the logical treatment of these cases, even though its limitations are clearly obvious. Because of these limitations, the problem of tooth restoration in its relation to occlusion is

the major problem of periodontia. These cases are, as a rule, best cared for by the specialist in prosthodontia in coöperation with the periodontist, who should keep the case under more or less careful observation after the restorative treatment has been completed, in order that the occlusion of the natural teeth may be checked up.

What type of restoration is best adapted to occlusal normalcy is a question which cannot be answered, as there is naturally a diversity of opinion.[35] Each case will have to be worked out as a problem by itself, if the best results are to be obtained. It is this type of case in which the periodontal tissues are traumatized because of tooth loss that is most difficult to classify. The fault may not have been primarily an abnormality of occlusion or weakness of the supporting tissues. Both, in a given case, may have been entirely normal at the time of tooth extraction; but later, when abnormalities in cusp relations developed as a result of excessive wear, and the teeth became overloaded, periodontal trauma was the logical outcome.

In the *second* group are to be found those cases in which the periodontal tissues are traumatized because of their incapacity to withstand the normal occlusal impact of mastication. Having been weakened by disease, the stress which in health contributed to their vigor, under conditions of disease, became their undoing. The fault was not in the magnitude of the force or in malocclusal relations, but in a subnormality of those tissues of the periodontium which resist occlusal stress. This being so, it follows that treatment logically consists in building up these tissues as far as that may be possible by the treatment of the existing periodontal disease, the correction of malocclusion and the restoration of lost teeth in order that they may resume their normal function of supporting the teeth under the stress of mastication. Since it is not always possible to

do this so completely as to bring about former conditions, it frequently becomes necessary to resort to the expedient of tooth-grinding in order to reduce the occlusal stress to the point at which it can be borne until such time as health shall have been reëstablished in the periodontal tissues. There are few, if any, cases in which all the teeth are present and in normal occlusion where occlusal stress develops through overload or cuspal incoördination, and none in which the grinding of teeth from third molar to third molar is ever justified. In those cases in which it is practiced, it should always be done with extreme caution, for as Johnson very aptly says: "Until we are in possession of more knowledge than we now have, the safest procedure in the treatment of periodontal lesions is the preservation of natural tooth forms and the study of the functional processes of the tissues which support them. Acute conditions and the limitations of our knowledge may necessitate the occasional grinding of a tooth. Nevertheless, the evidence is clear enough that the functional processes primarily at fault are periodontal, not occlusal. Grinding a tooth in one part of the arch involves adaptive processes in other regions. Hence, in the presence of periodontal lesions, indicative as they are of impaired function of the tissue, the probability, when grinding, of initiating lesions elsewhere should not be overlooked. . . . The biologic significance of tooth forms is as vital as the structural constitution of periodontal tissue. They are functionally interdependent. Periodontal tissues, being more responsive to the vicissitudes of life, reflect them more readily than do tooth crowns. Thus, periodontal tissue is more often the seat of the primary factor in pathologic lesions than is the crown of the tooth in the function of occlusion. In the vast majority of cases, occlusion is of secondary importance in the development of periodontal lesions." [22]

REFERENCES

[1] Best, E. S., 1927, "Examination and Case Records," *Journal American Dental Association,* vol. 14, p. 890.

[2] Box, H. K., 1921, "The Evolution of the Periodontal Pus Pocket," *Journal American Dental Association,* vol. 8, p. 599.

[3] Box, H. K., 1924, "Studies in Periodontal Pathology," *Canadian Dental Research Foundation,* Bulletin 7.

[4] Box, H. K., 1928, "Treatment of the Periodontal Pocket," *Canadian Dental Research Foundation,* Bulletin 10.

[5] Bunting, R. W., 1923, "The Diagnosis of Periodontal Diseases," *Journal American Dental Association,* vol. 8, p. 647.

[6] Fleming, W. C., 1926, "Localization of Pyorrheal Involvments," *Dental Cosmos,* vol. 68, p. 538.

[7] Fordyce, J. A., and MacKee, G. M., 1926, "Diseases of the Oral Mucosa," Archives of Dermatology and Syphilogy, vol. 13, p. 601.

[8] Garvin, M. H., 1927, "The Principles of Practice in Periodontia with Special Reference to the Sequence of Steps in Treatment," *Journal American Dental Association,* vol. 14, p. 905.

[9] Gottlieb, B., 1927, "Tissue Changes in Pyorrhea," *Journal American Dental Association,* vol. 14, p. 2178.

[10] Gottlieb, B., 1926, "What is a Normal Pocket?" *Journal American Dental Association,* vol. 13, p. 1747.

[11] Gottlieb, B., 1928 "Traumatic Occlusion," *Journal American Dental Association,* vol. 14, p. 1276.

[12] Hartzell, T. B., 1925, "Etiology of Pyorrhea Alveolaris with a Simplified Treatment," *Journal American Dental Association,* vol. 12, p. 1452.

[13] Hartzell, T. B., 1927, "Important Factors in the Etiology and Control of Periodontoclasia," *Journal American Dental Association,* vol. 14, p. 899.

[14] Hartzell, T. B., 1927, "Chronic Infections of the Mouth and Teeth and the Tissues Investing Them," *Journal American Dental Association,* vol. 14, p. 1253.

[15] Hartzell, T. B., 1916, "Some Evidence of the Importance of the Dental Path as a Source of Serious Localized and General Infection," *Journal American Dental Association,* vol. 3, p. 172.

[16] Hatfield, H. K., 1925, "A Study of Traumatic Occlusion from the Orthodontic Point of View," *Journal American Dental Association,* vol. 12, p. 1437.

[17] Hayden, Gillette, 1917, "Periodontal Inflammations Resulting from Accumulations of Foreign Substances upon the Teeth," *Journal American Dental Association,* vol. 4, p. 3.

[18] Hellman, M., 1921, "Variations in Occlusion," *Dental Cosmos,* vol. 63, p. 608.

[19] Hirschfeld, I., 1926, "Further Studies of Skulls in Relation to Periodontal Diseases," *Journal American Dental Association,* vol. 13, p. 958.

[20] James, A. F., 1927, "Aims and Results of Periodontal Treatment," *Journal American Dental Association,* vol. 14, p. 903.

[21] Johnson, A. L., 1923, "Basic Principles of Orthodontia," *Dental Cosmos,* vol. 56, p. 509.

[22] Johnson, A. L., 1928, "Occlusion an Etiologic Factor in Periodontal Lesions," *Journal American Dental Association,* vol. 15, pp. 510, 511.

[23] Jones, M. R., and Simonton, F. V., 1928, "Mineral Metabolism in Relation to Alveolar Atrophy in Dogs," *Journal American Dental Association,* vol. 15, p. 881.

[24] Leonard, H. J., 1929, "Periodontoclasia: Cause and Treatment," *Journal American Dental Association,* vol. 16, p. 629.

[25] MacMillan, H. W., 1928, "The Clinical Significance of Disuse Atrophy of the Alveolar Process," *Journal American Dental Association,* vol. 14, p. 699.

[26] MacMillan, H. W., 1928, "Nonuse in the Development and Resistance of the Alveolar Process," *Journal American Dental Association,* vol. 15, p. 511.

[27] Merritt, A. H., 1927, "A Résumé of Present-Day Knowledge Regarding the Pathology, Etiology and Treatment of Suppurative Periodontoclasia," *Dental Items of Interest,* vol. 49, p. 77.

[28] Mitchell, C. G., 1926, "Pyorrhea and Diet," *Dental Cosmos,* vol. 68, p. 195.

[29] Orban, B., 1927, "Nutrition and Teeth," *Journal American Dental Association,* vol. 14, p. 1619.

[30] Pollia, J. A., 1928, "Fundamental Principles of Alveolo-Dental Radiology," *Dental Items of Interest,* vol. 50, pp. 784, 786.

[31] Simonton, F. V., 1925, "A Conservative Method for the Treatment of Pyorrhea," *Journal American Dental Association,* vol. 12, p. 197.

[32] Simonton, F. V., 1925, "Examination of the Mouth with Special Reference to Pyorrhea," *Journal American Dental Association,* vol. 12, p. 287.

[33] Simonton, G. W., 1925, "The Treatment of Pyorrhea by Surgical Exposure of the Involved Tissues," *Journal American Dental Association,* vol. 12, p. 1452.

[34] Smith, Julian, 1927, "The Principles of Practice in Periodontia—Root Surgery," *Journal American Dental Association,* vol. 14, p. 909.

[35] Smith, Julian, 1928, "Operative Procedures in Periodontia," *Journal American Dental Association,* vol. 15, p. 1317.

[36] Stillman, P. R., and McCall, J. O., 1921, "Periodontology—Etiology, Diagnosis and Treatment," *Journal Dental Research,* vol. 3, p. 73.

[37] Stillman, P. R., 1917, "Prophylaxis of Pyorrhea," *Journal American Dental Association,* vol. 4, p. 156.

[38] Tishler, B., 1926, "Present-Day Treatment of Suppurative Periodontoclasia," *Dental Items of Interest,* vol. 48, p. 579.

[39] Kirkland, Olin, 1925, "Oral Hygiene and Periodontia in General Practice," *Journal American Dental Association,* vol. 12, p. 1217.

CHAPTER IX

INSTRUMENTATION

Instrumentation plays a part of such importance in the treatment of periodontoclasia as to justify its separate consideration, even though this may involve some repetition. Indeed, instrumentation is the *only* treatment, and the sooner this is learned by the rank and file of the profession the better it will be for all concerned. Since therapeutics have always played so large a part in the treatment of disease, it is perhaps not unnatural to think in these terms regarding the treatment of periodontoclasia. Especially is this true of those who have not made an intimate study of its etiology and pathology. One has only to understand these, however, in order to realize how fallacious and illogical are all therapeutic measures in the treatment of periodontoclasia. This is not to say that they may not in some instances possess some supplementary value, but in no case are they ever anything more than this, and then only as adjuvants to instrumentation.

Instrumentation is so essential to the treatment of periodontoclasia that, when skillfully performed, there are few cases that require any other treatment, except such attention to the occlusion as may be necessary to correct it. When it is inefficiently done, no other treatment, whether it be vaccines, drugs, or gum stimulation, will suffice to remedy this defect. The belief that the teeth can be "scaled" and the pockets injected with some unknown and miraculous mixture is one that has been fostered by the makers of these secret and unscientific preparations. The

author recently received a circular letter saying that if he would "scale the teeth and inject this preparation into the pockets" there would be "no pus after three or four such treatments." He replied that since he was able to accomplish this very desirable object in one treatment by instrumentation, and without the use of any drugs whatsoever, he was unable to see any virtue in the proposed "remarkable remedy." It is not at all improbable that, if the whole truth were known, it would be found that the injection into the pockets of any preparation hinders, rather than helps, by actually interfering with Nature's processes of repair. Until more is known regarding the laws governing chemotaxis, one should exercise great caution in introducing any drug into a periodontal pocket.

THE APPLICATION OF INSTRUMENTS

Having completed the examination, which, as pointed out, should always include a complete radiographic survey, the next step in order is instrumentation. The only exception to this sequence is in those cases where a few individual teeth, obviously weakened by the loss of alveolar support, are being traumatized in the act of mastication. These should be ground to relieve them of excessive pressure. Any adjustment of the occlusion more than this should wait on instrumentation.[2] In those cases in which the hygiene of the mouth is bad, and where there are large accumulations of salivary calculus, a preliminary cleaning of the teeth should precede instrumentation. With these possible complications out of the way, the case is ready to begin subgingival curettage. This should be done in an orderly manner. That is, one should begin at a definite place in the mouth, and from that point proceed from tooth to tooth around the entire mouth, completing the treatment of each tooth or group of teeth before passing on to others,

an exception being made of those cases only in which there are individual teeth causing pain or otherwise giving trouble.

THE AUTHOR'S TECHNIQUE

A satisfactory plan of operation is to begin on the lower right side, and treat each individual tooth in turn until all the teeth of the lower jaw have been finished, after which one may return to the right side and repeat in the upper jaw the same procedure employed in the mandible. While the order of treatment will vary with each operator, certain principles should be observed in the use and application of instruments. Perhaps this can best be illustrated by outlining in some details the methods employed by the author, though, in doing so, he is not insisting that another shall follow the same procedure. The thing that is important is, that the operator should have in mind before he picks up a single instrument the order in which he is to proceed, and the different instruments that will be needed as he progresses step by step. Also, that as far as possible, these instruments be used in sequence, and the work each is designed to do be finished before it is replaced by another. This is in the interest of efficiency, rather than that one may not accomplish results, even though in achieving them many false motions are made. One should always bear in mind that the most skillful worker in any field is he who achieves the maximum of results with the minimum expenditure of time and effort. Not only should one practice efficiency for efficiency's sake, but he should do so because, in the treatment of periodontoclasia, such methods are less painful, at the same time that they are less costly. Inefficiency and bad technique explain practically all the pain associated with the treatment of the average case, and, in some instances, the pain of excessive fees also.

THE USE OF CURETTES

Since the author is predisposed in favor of curettes, it is largely their use that will be described. He favors them because they are more delicate in their construction, and therefore convey to the fingers of the operator a keener sense of touch than is possible by any other type of instrument known to him. In subgingival curettage this is highly important, inasmuch as the field of operation is never visible. A further reason is that their use causes less trauma to the tissues operated upon, and therefore less operative

FIG. 39.—Instrument No. 18 being brought to bear on the distal surfaces of lower teeth from lingual side; second and third fingers resting on lower incisors.

and postoperative pain. Files are recommended by some operators and in certain places may be used to advantage, though it is doubtful whether they accomplish anything which cannot be done with more accuracy of technique with curettes or planes. This is not to say that they should not be used, but to point out that their use is not essential to success.

With number 18 of the McCall curettes in hand, insert it in the pocket and bring it to bear on the distal surface of the root of the lower right third molar (or whichever happens to be the last tooth), approaching it from the lingual side. (Fig. 39.) With a pull motion, curette this surface laterally; that is, buccolingually, as far as the

instrument can conveniently be brought to bear. This is in the nature of a smoothing and polishing, rather than a scaling, operation, though any tartar which may be present should be removed. As the surface becomes smooth under the repeated action of the instrument, it is moved slowly up and down from the bottom to the top of the pocket, in order that one may feel more accurately the condition of the root. From time to time, the instrument should be lifted away from the root surface to make sure that what had been thought to be the bottom of the pocket is not, in reality, an obstructive piece of tartar. It should not be forgotten, in this operation, that the entire surface of the root, as far as it is involved by the pocket, is a septic surface, covered by the remnants of the necrotic pericementum, serumal tartar, bacteria, etc., and that this must be completely removed as a prerequisite to successful treatment. The idea that the removal of tartar is all that is necessary is a fallacy, and unfortunately, a common one.

FIG. 40.—No. 17 being applied to the distal surfaces from the buccal side. Fingers of right hand resting on those of the left.

Having completed the curettement of the distal surface of the tooth root on which treatment was begun, proceed to the same surface of the tooth next in front and curette it, also using instrument number 18. In the interest of efficiency and expedition, no instrument should be laid aside until all the teeth to be treated in the time reserved have been completed. In other words, if it is believed that

five teeth can be completed in one operation, the distal surfaces of all five should be curetted and finished with number 18 before laying it down and beginning with number 17, the instrument next in order. The same rule applies to the use of all instruments. Complete the work each is designed to do before proceeding to the next. In this way many unnecessary motions are avoided and time, which is the cost element in all dental operations, is conserved.

Number 17 of the McCall set is next brought to bear on the distal surface of the same tooth, approaching it from the buccal side. (Fig. 40.) With this instrument, that portion of the distal surface not reached by number 18 is curetted in the same way. With these two instruments all of the distal surfaces are curetted, as are also the bucco-lingual angles.

Fig. 41.—Instrument No. 3 as it engages the mesial surfaces of the teeth, lower left side, lingual approach. No. 4 should be used from buccal side. Right hand poised on left.

In all operative work in periodontia, it is essential that the hand holding the instrument be firmly fixed at some point. In the use of instrument number 18 as above outlined, this is most conveniently done by resting the second and third fingers on the lower incisors. In using number 17 as it engages the distal surfaces of the teeth on the lower right side from the buccal angle, the fingers of the right hand as a rule rest most advantageously on the fingers of the left hand as they stabilize the lower jaw and keep the cheek and tongue out of the operative field. In much of

the work done on the mandible, this latter position will be found to be the most satisfactory way in which to firmly fix the right hand. (Fig. 41.) Once this way of stabilizing it has been learned, it will, wherever possible, be given preference over every other method as affording the most satisfactory rest obtainable. Having finished the treatment of the distal surfaces of the teeth on the right side to be

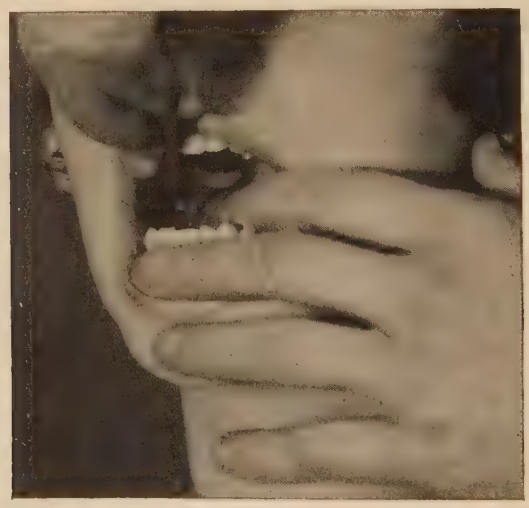


Fig. 42.—No. 18 curetting mesial surface lower right side from lingual side. Nos. 4 and 14 may also be used. Fingers of right hand resting on upper incisors and cuspid as the operator stands behind and to the left of patient.

operated upon at that sitting, attention is next given to the mesial surfaces. Standing behind and to the left of the patient, the convex angle of instrument number 18 is brought to bear on the mesial surface of the roots of each tooth from the lingual side, resting the hand meanwhile on the incisor teeth of the upper jaw. (Fig. 42.) Numbers 3 and 4, 13 and 14 of the Younger set, can also be used to excellent advantage on the mesial surfaces of all teeth, either as supplemental to numbers 17 and 18, or

when there is little or no tartar present, independent of their use. They can be brought to bear on all mesial sur-

FIG. 43.—Nos. 3 or 13 as they are brought to bear on the mesial surfaces of all teeth on lower right side. Right hand resting on the fingers of the left as they hold the lips and cheeks out of the operative field.

faces with great efficiency. (Figs. 43, 44.) With these six instruments, the mesial and distal surfaces of most of the

FIG. 44.—Instrument No. 13 curetting the mesial surface of a lower incisor. No. 14 would apply to the same surface on the lower left side. Right hand seated on fingers of left.

teeth can be curetted very satisfactorily, leaving only the buccal and lingual surfaces. In many instances these also

can be operated upon with these same curettes, but if the pockets are deep, it is often most satisfactorily accomplished with a plane instrument such as numbers 13 and 17 of the Towner set. In addition to these numbers, they have the letters B and M, indicating in this way their applicability to bicuspids or molars. In many instances they can be used interchangeably, as is true of most instruments used in periodontia. The planes are carried to the bottom of the pocket and the entire surface between the mesio-

FIG. 45.—No. 13B of Towner's planes as it is applied to the lingual surfaces of lower incisors. Note right hand resting securely on left as the fingers divide to protect lips and tongue.

distal angles not covered by the curettes is operated upon, using a pull motion, though occasionally a push stroke is employed in dislodging heavy deposits of tartar. Numbers 11 and 15 of these instruments may often be used to advantage on the mesial and distal surfaces of teeth as supplemental to the curettes. The teeth on the left side are treated in the same way, though the number of the instrument is reversed. Deep pockets involving the labial or lingual surfaces of teeth are most satisfactorily curetted with a plane-shaped instrument, as the blades of these

instruments are more easily brought to bear in these cases than are those of the curettes. Several of the Towner periodontomes are admirably adapted to meet this situation. (Fig. 45.)

Fig. 46.—No. 17 as it is applied to the distal surfaces of teeth, upper right side, buccal approach. Right hand resting on left.

In some respects the upper teeth are more difficult to operate upon than are those of the lower jaw. They are less accessible and direct vision is not always possible. In operating upon the upper teeth, begin also on the right side, bringing to bear the convex surface of number 17 on the distal surface of the teeth being operated upon, approaching them from the buccal side. (Fig. 46.) The concave angle of the same instrument is used in curetting the distal surfaces from the lingual side, while its convex angle is well adapted to curetting the mesial surface from the lingual approach. The convex angle of instrument number 18 is applicable to the mesial surfaces of all teeth on the upper right side when approached from their buccal aspect. Numbers 3 and 4, 13 and 14 of the Younger curettes—are admirably suited to the curettement of

Fig. 47.—Curetting mesial surfaces with instruments Nos. 4 and 14, upper right buccal side.

the mesial surfaces of all teeth and should always be used after the coarser deposits have been removed by the heavier instruments, numbers 17 and 18. (Fig. 47.) These instruments (Nos. 3, 4, 13, 14), as made by Charles Grafrath, 556 West 48th Street, New York City, are very delicate and for that reason are to be preferred to those made by some manufacturers. They make possible a sense of touch in their use possessed by no other instruments known to the author. Wherever they can be brought to bear they are the instruments of choice after the grosser deposits have been removed. Unfortunately, they are not applicable to the distal surfaces of posterior teeth. As in operating in the lower jaw, the right should always be firmly seated. This is most often done by resting it somewhere on the lower teeth or chin. (Fig. 48.) In operating on certain of the upper teeth, the hand may be poised on the fingers of the left hand, though there are not so many opportunities for doing this as when operating on the teeth of the mandible. Occasionally, as when operating on the mesiolingual surfaces of the teeth of the upper jaw, left side, the thumb of the left hand is brought to bear on the instrument in such a way as to assist greatly in the operation. Especially helpful is this joint use of the hands (which is easier demonstrated than described), when using numbers 3 and 4 and 13 and 14 of the Younger set. (Fig. 49.)

Fig. 48.—No. 18 as applied to distal surfaces, upper left side. Right hand resting on patient's chin.

The application of all instruments to the left side of the

mouth is exactly the reverse of that on the right side. For example, the distal surfaces would be approached from the buccolabial side with number 18 instead of number 17, a fact more or less obvious to anyone versed in the use of instruments, in "rights and lefts."

FIG. 49.—The application of instruments Nos. 4 or 14 to mesial surfaces lingual side. Right hand resting on lower incisors. Note the thumb of the left hand as it is brought to bear on the instrument. It is of great assistance in bringing the blade to bear on the root surface.

TREATMENT OF INNER WALL OF POCKETS

It will be remembered that in some cases there is a tendency for the epithelium covering the external surfaces of the gingivæ to extend into and line the inner wall of periodontal pockets, in this way presenting a more or less healed surface. This naturally needs to be broken up in order to encourage reattachment. To do this, some advocate the use of certain drugs, or combination of drugs, such as the sodium sulphide mixture of McCall's. This latter is composed of the following:

Sodium sulphide	70 grams
Sodium carbonate	20 grams
Water	1 ounce

This is applied by rolling small ropes of absorbent cotton and saturating them with the mixture full strength, packing them in the pocket, and allowing them to remain ten minutes. They should then be removed, the pocket sprayed out, and the application repeated, if necessary. This prepa-

ration acts as a depilatory and is as well adapted to its purpose as anything of a therapeutic nature could be. There are few, if any cases, however, in which instrumentation will not serve the purpose of breaking up this lining membrane which has the advantage of being at once logical and simple. As already indicated, the use of drugs in periodontal pockets is to be avoided whenever possible.

There are also pockets, the inner walls of which present a more or less unhealthy hypertrophic surface. This condition is most often met with in pockets that are deep and wide open, so to speak, and in which there is considerable congestion of the surrounding tissues. It is desirable that this be broken up as a part of subgingival operation, and it can usually be done in connection with the instrumentation designed to curette the root surface. In other words, this can and will be done in the process of root curettement, without making a special effort to curette the gingival wall of the pocket. In shallow pockets, and those in which the surrounding gingivæ and gums show little congestion, this hypertrophied tissue is less likely to be present. This is in part due to the fact that in such cases there is little or no serumal tartar on the root surface. Heavy deposits of tartar on a tooth root seem to have the property of irritating the inner wall of the pocket, thus causing it to react by laying down a lining of granulating tissue. This tissue is often referred to as "granulomatous tissue," with the implication that it is comparable to the periapical granuloma, and that a special effort should be made in the treatment of these cases to remove it. This is a mistake. It is simply Nature's reaction to irritation, which in these cases is mechanical, bacterial and chemical. Clinical experience has repeatedly shown that when these irri-

tating factors are removed by subgingival curettage with such scarifying of the inner walls of the pocket as would naturally take place in this procedure, no other treatment is necessary. To cut away these tissues or to make a special operation of curetting their inner surface is usually unnecessary and therefore unwise. Regarding the nature of this tissue and its treatment, Berger says: "Much stress is placed on the importance of the removal of the granulation tissue adhering to the inner surface of the gum that forms the pocket, and often penetrates the excavated alveolar process. This tissue is classed by some with the granuloma that occurs at the ends of infected roots. I doubt the accuracy of this comparison. Clinically, these tissues appear to be sickly, soft, poorly organized, very friable and loose in texture. They do not appear to form an organized entity as does the granuloma. Histologic examination shows that they are here and there pervaded by epithelial chains. These may be derived from the surface epithelium, but more likely arise from the epithelium of the gingival crevice or from the epithelial chains of the periodontal membrane.

"This type of tissue is often found to develop about necrosed bone or in chronic suppurative areas. It is a common clinical observation that, *with the removal of the irritating factor,* the sickly tissue proliferation has a tendency to *become resorbed. This is true whether it develops about the end of a root, about a portion of necrosed bone, or in an ulcerative pyorrhea pocket.* Some of the tissue breaks down and is transformed into pus; some may undergo autolysis, and a considerable portion may be destroyed by the activities of the phagocytic cells. To those who have had the opportunity to treat and closely observe cases of necrosis, this is not an unfamiliar phe-

nomenon. In these cases we often see retrograde changes and the resorption of such tissue proliferation on the *removal of the etiologic factors.*" [1]. (Italics not in original.)

THE SENSE OF TOUCH

Still another factor to be considered in the successful use of instruments in the treatment of periodontal lesions is a delicate sense of touch. It is the possession of this faculty by the periodontist that makes the difference between an artisan and an artist. With some, it is instinctive; with others, it may be absent altogether. To a considerable extent, it can be acquired, though the stories of painful treatment one often hears from patients might lead one to believe that this is not true. Its possession is the first requisite to success in periodontia. One may observe every other principle in instrumentation and still fall short of complete success by the absence of this one faculty. It is something that should be cultivated by every dentist, for there can be no doubt that a measure of success will attend all efforts to acquire it. Once learned, most of the failures and pains of instrumentation will automatically cease to exist.

SUMMARY

In all operative procedures in periodontia, it is inevitable that there should be individual differences in technique. No two will operate in exactly the same way. And within certain limits, it is not necessary that they should. It is principles rather than details representing individual preferences that should be observed. These are:

First: System in operative procedure; Second: Sequence in the use of instruments; Third: Fixity of operating hand; Fourth: Deftness of touch. When these have been learned, and they apply with equal force to the postoperative care

of cases, one need not worry about the details; they will take care of themselves.

REFERENCES

[1] Berger, A., 1927, "The Surgical Treatment of Periodontoclasia," *Journal American Dental Association,* vol. 14, p. 1019.

[2] McNulty, T. P., 1929, "Treatment of Tissues Supporting the Teeth," *Dental Digest,* vol. 35, p. 367.

CHAPTER X

THE SURGICAL TREATMENT OF PERIODONTOCLASIA

Having considered at some length the treatment of periodontoclasia by the conservative method of subgingival curettage, it becomes necessary to discuss its surgical treatment. Since it is the nature of this disease to destroy the osseous support of the teeth by the resorption of the alveolar process, in this way creating pockets about the teeth, it naturally follows that the depth of these pockets will, other things being equal, depend upon the amount of bone resorption. In no case does the retraction of the gingivæ keep pace with the bone loss, though there is frequently some variation in this respect. As a rule, gum recession is not marked when compared with the loss of bone. This means that the more advanced the bone resorption, the deeper will the pockets be. For this reason, it may not be possible to pass an instrument into the gingival orifice of the pocket that will reach its ultimate boundaries. This is especially true of those cases in which the gum tissues are firm and the gingival entrance to the pockets constricted. There are instances in which these tissues are inflamed and more or less relaxed, making it possible to do, by subgingival curettement, what might not be possible in another case no further advanced.

DESIRABILITY OF CONSERVATIVE TREATMENT

It will thus be evident that the more advanced the case, the less probable is it that it can be successfully treated

by the more conservative method of subgingival curettement. Nevertheless, the greater the skill and experience of the operator, the more resourceful does he become in the treatment of these cases, and the less often does he find it necessary to resort to the more drastic operations of surgery. It should be the aim of every worker in this field (and that should include every dentist) to so perfect his technique that there will be few instances in which he will find it necessary to take refuge in surgery, and none in which the entire mouth need be so treated. All will agree that where equally good results can be obtained by conservative methods, they outweigh in value those of surgery.[1]

Since the progress of periodontoclasia is rarely uniform throughout the mouth, it is a matter of common observation that there may be some teeth seriously involved, while others in the same mouth are only slightly or not at all involved. If the case under treatment be one of long neglect, there may be teeth that will have to be extracted, and others so far advanced that they cannot be successfully treated, as they at one time could have been, by the more satisfactory method of subgingival curettage. It is these cases in which there are teeth that have passed beyond the bounds of conservative treatment that justify resort to surgical measures, for the only alternative is extraction.

TYPES OF SURGICAL OPERATIONS

There are three more or less distinct types of operations for the treatment of these cases:

First: Gum resection, sometimes referred to as gingivectomy, by which the gum forming the pocket is excised to or below the level of the involved alveolar process, and the root exposed.[3]

Second: A flap operation, consisting of two vertical and parallel incisions, usually limited to the buccolabial sur-

faces, the laying back of the gum flap and the exposure of the field to be operated upon.[5]

Third: In those cases in which there is a group of several teeth to be operated upon, the septal gingivæ between the individual teeth are incised to the floor of the pocket, the buccolingual gums are forced back in such a way as to gain direct access to the field of operation. Following the operation, the separated gums are brought back into place and sutured between each tooth.

These are all done, as a rule, under local anesthesia, though there are a few operators who hospitalize their patients, give them a general anesthetic, and resect the gums of the entire mouth at one operation.[3] In the case of a local anesthetic, where there are several teeth to be operated upon on one side of the jaw, a conductive anesthesia is desirable. In less extensive cases, an infiltration may be used and has the advantage of assisting in the control of the hemorrhage.

In all surgical operations, the treatment of the root surfaces is the same as in subgingival curettage, though it is naturally more easily done because more accessible. Usually a more radical curettement of the alveolar border is practiced than in subgingival treatment. This is partly to assist in its subsequent covering by the gum tissues, and by making the line of the border more symmetrical, to minimize disfigurement.

GUM RESECTION

Because of its disfiguring effect, gum resection should, as a rule, be limited to the posterior part of the mouth or to the lingual surfaces of teeth. Its most logical place is about those teeth in which the pocket and bone loss involve the bifurcations of molars and where in consequence it is difficult to control reinfection; also, in those cases in which

there is extensive bone loss, more or less limited to the interproximal spaces and creating a deep pocket by the presence of the loose and poorly nourished septal gingiva. Frequently also as a result of the extraction of third molars and the absorption of the alveolar process distal to the second molar, a deep and narrow pocket is formed that is difficult to manage. In all these cases, and especially in the molar region where more conservative measures have failed, gum resection may be successfully employed. It must be said, however, that it is the most radical of all forms of periodontal surgery and, in some respects, the least satisfactory.

THE "FLAP OPERATION"

The "flap operation," also called the Robicsek operation [1] is more or less limited to the buccolabial surfaces of the gums, and has the advantage over the resection method of being less disfiguring. It is most often practiced in the incisor region where there are several deep, narrow pockets. Two vertical incisions extending from the depth of the pocket to the gingival border are made, the septal gingivæ are divided and the labial flap laid back exposing the anterior surfaces of the four or five teeth to be operated upon. Access can usually be had to the lingual surfaces without the vertical incisions. The root surfaces are curetted, any unhealthy tissue that may be present is generally removed, and the flap brought back into place and sutured. The sutures should be allowed to remain for three or four days or until union has taken place. The same operation is also applicable to the bicuspid and first molar regions, especially in the upper jaw. In those cases also where there is a deep, narrow pocket about an individual tooth, a single incision parallel with the root can be made, extending from the border of the alveolar process to the

orifice, dividing the pocket midway between its anteroposterior boundaries. In this way direct access to the pocket is gained. After the root surface has been curetted, the divided tissues are brought into apposition, where they will usually reunite without suturing. In such cases there is occasionally a slight recession following healing, though this is never sufficient to cause disfigurement.

DIVIDING OF THE SEPTAL GINGIVÆ

In those cases where there are several teeth in a row with deep pockets, the septal gingivæ are divided and the more or less loose buccolingual gums are drawn back, exposing in this way the area to be operated upon. It is especially applicable to the bicuspid and molar regions. At the conclusion of the operation, the buccolingual gums are brought into apposition and sutured at the point of incision between each tooth. The operation is much the same as the flap operation without the vertical incision, and yields the maximum of results with a minimum of disfigurement. The gum recession following this operation is less evident than in either of the other types of operations, though always greater than that attending subgingival curettage. The postoperative pain and discomfort in all surgical cases are also greater than that following subgingival treatment, as might be expected.

Fig. 50.—Chronic gingivitis complicated by badly fitted crowns.

SUMMARY

These operations all have their place in periodontia, and when practiced with discrimination make possible the sav-

ing of teeth that would otherwise have to be extracted. The claim is made for them that they require less time than treatment by subgingival curettage, and that there is less danger from reinfection, both of which are probably true in extensive cases. The objection urged against them is postoperative pain, disfigurement, sensitiveness of exposed surfaces, increased difficulty in keeping the mouth and teeth clean, etc. These are all valid objections and need to be considered in every case. To control sensitivity of exposed root surfaces, Dr. A. W. Ward,[4] of San Francisco, recommends the use of a cement-like substance to be packed around and between the teeth immediately after operation, and which may be allowed to remain from a few days to several weeks. It is available under the trade name of "Wonderpak" and according to Dr. Ward is composed of the following:

Powder	Zinc Oxide
	Magnesium oxide
	Resin
Liquid	Oil of Cloves
	Olive Oil
	Leptinol
	Scarlet Red

The liquid and powder are mixed together into a stiff paste. As it sets slowly ample time is afforded for its manipulation. It is only slightly adhesive, and when applied to individual teeth it is apt to be dislodged. It is not necessary to dry the surfaces to which the cement is to be applied.

REFERENCES

[1] Berger, A., 1927, "The Surgical Treatment of Periodontoclasia," *Journal American Dental Association,* vol. 14, p. 1013.

[2] Towner, J. D., 1922, "The Surgical Treatment of Periodontal Lesions," *Journal American Dental Association,* vol. 9, p. 706.

[3] Ward, A. W., 1928, "The Surgical Eradication of Pyorrhea," *Journal American Dental Association,* vol. 15, p. 2146.

[4] Ward, A. W., 1929, "Post-operative Care in the Surgical Treatment of Pyorrhea," *Journal American Dental Association,* vol. 16, p. 635.

[5] Zentler, A., 1920, "The Surgical Treatment of Suppurative Gingivitis, so-called Pyorrhea," *Journal Dental Research,* vol. 2 p. xvii.

CHAPTER XI

RESTORATIVE TREATMENT

At first thought it might seem inappropriate that the treatment of periodontoclasia should include the restoration of lost teeth. Yet the logic of such treatment will be self-evident when one reflects upon how large a part tooth loss plays in the breaking-down of the periodontal tissues. This is due to the changes which occur in the positions of teeth, causing malocclusion, open contact points, and, in many instances, excessive occlusal stress from overloading. In the treatment of periodontoclasia, nothing is more prejudicial to a favorable prognosis than tooth loss. It almost invariably creates an unfavorable prognosis just in proportion to the number of missing teeth. How to correct this condition so as to restore masticating efficiency and occlusal balance, without at the same time injuring the remaining teeth, is one of the most difficult problems in periodontia. In many instances it is a problem that cannot be solved with more than partial success. The real issue in all partial restorations, and especially those involving the molars, is not primarily masticating efficiency, but how, by means of such restorations, excessive occlusal stress can be prevented on the remaining natural teeth. There can be no fixed rule for the treatment of these cases.

Having restored to health the tissues of the periodontium through treatment, the case should, as a rule, be placed in the care of a competent prosthodontist for the restorative treatment. When this is done, there should be the closest

coöperation between the periodontist and prosthodontist. If the case be under the care of a general practitioner of dentistry who is competent to give both the periodontic and prosthetic treatment, so much the better. All cases in which prosthetic treatment is necessary require more watchful postoperative care, because of the increased difficulty in such cases of maintaining normal conditions. This means more frequent visits for prophylactic treatments, at which times particular attention should be given to the occlusion.

RESTORATION OF TOOTH FORM

Under the head of restorative treatment must also be included restoration of tooth form, normal contact relations, etc.[2] By restoration of tooth form is meant the reproduction of cusps in those cases where through caries or fracture these have been lost, with possibly the lingual or buccal surfaces also. The flat filling so often seen in these cases, where no attempt whatever is made to restore the original contours of the tooth, should not be tolerated. By the use of gold and porcelain, these abnormalities in tooth form are so easily and satisfactorily restored that there can be no excuse, except possibly an economic one, for not doing so. This aspect of the problem should always receive consideration and the patient should be advised in the matter.

RESTORATION OF PROXIMAL CONTACTS

The restoration of proximal contacts, where these have been lost through caries or tooth extraction, is of even greater importance, for in all such cases more or less trauma of the septal gingivæ is produced by food impaction. As a rule, this is most satisfactorily accomplished by the use of gold inlays. Porcelain may be used in cases where the æsthetic must be given first consideration. Plastic fillings

rarely give wholly satisfactory results. Unfortunately in this, as in every other field of endeavor, compromises for one reason or another may have to be made, and the second best be given the right of way. Under such conditions the operator should do everything possible to correct the situation, and the patient should share with him responsibility for the results. Fortunately for all concerned, Nature has a way of accepting the second best and continuing to function with more or less satisfaction. If this were not so, one might well despair.

THE INTERDEPENDENCE OF PERIODONTIA

It will be seen from the foregoing that periodontia is inextricably bound up with the general practice of dentistry, and that if it is to yield its full measure of service, it can do so only as it coöperates with every other branch of dental practice. It would be fortunate, indeed, if every one were versatile enough to comprehend within himself the varied and exacting requirements of modern dentistry. But since this is only infrequently the case, the individual who limits himself strictly to periodontia should seek the assistance of those best qualified to coöperate with him in order that his patients may receive the maximum of service. One of the best ways to accomplish this is through group practice, where several work together as a unit in the general practice of dentistry. It has the advantage of closer coöperation than is possible where the patient is sent out of the office and is always more satisfactory to all concerned.

In whatever way, however, the situation is met, only the patient's interests should be considered. If these are better conserved by referring him out of the office, this should be done. One must never lose sight of the fact that the patient and his interests are to be given first consideration under all circumstances.

REFERENCES

[1] Schott, C. H., 1928, "One Piece Cast Removable Appliances," *Journal American Dental Association,* vol. 15, p. 1299.

[2] Spalding, E. B., 1918, "Restoration of Portions of Teeth," *Journal Allied Dental Societies,* vol. 13, p. 123.

CHAPTER XII

INSTRUMENTS

The type of instruments used in the treatment of periodontoclasia is a matter of such importance that it should be carefully considered. Like everything else having to do with man's needs, they have evolved in direct ratio to his recognition of that need, and as his experience in their use has increased. When Riggs began his study and treatment of what was then known as "scurvy of the gums" there were no instruments available for its treatment. He was under the necessity of designing his own, which he did. These were six in number, and as compared with those in more or less general use at present, they were coarse, clumsy and relatively inefficient. That he achieved a measure of success in their use there can be no doubt, though one wonders how he did so. What they lacked in delicacy of touch, Dr. Riggs made up for by an added contribution of "elbow grease." The operation of subgingival curettage as performed by him was an ordeal such as few patients of the present would endure. On this point, the late Dr. Levi C. Taylor, who was at one time associated with Dr. Riggs, in a personal communication to the author written several years ago, says, "Dr. Riggs was very rough and seemingly harsh, but for all that he produced the best results I have ever seen. He would place his patients in some instances under chloroform far enough to do his work satisfactorily." The report is sometimes made that he was an advocate of and practiced the surgical removal of the gums in the treatment of "pyorrhea alveolaris." This is not so. In a report of an address made by him before the Southern Dental

Association at Baltimore, August 9, 1882, and published in the *Dental Cosmos,* Vol. 24, p. 527, it is said: "The reason the profession are not familiar with the treatment of this disease is, they fail to recognize it until it reaches its third or fourth stages, and then they treat it by depletion and therapeutic remedies. Some treat it by stippling in acids underneath the gum, thinking thereby to dissolve away not only the tartar, but the necrosed bone. Another takes off patches of diseased tissue, and another a strip of gum from wisdom tooth to wisdom tooth. This treatment he could only characterize as simply barbarous." It is evident, therefore, that the pain inflicted by him in operating, as referred to by Dr. Taylor, was due not to operations on the gums themselves, but to the crudeness of his instruments and the rather heroic way in which he made use of them.

From these crude beginnings in instrument-making by Dr. Riggs, they have evolved into the large number of scientifically designed and carefully tempered instruments of the present. Not only have their numbers greatly increased, but they have undergone great refinement in design. The present-day operator, in his treatment of periodontal lesions, no longer depends upon the strength of his "good right arm" or the patient's powers of endurance as Riggs seems to have done, but upon his sense of touch as this is conveyed to him from the unseen point of a sharp and exceedingly delicate instrument as it engages the root surface within a periodontal pocket.

THE EVOLUTION OF INSTRUMENTS

As might be expected in this evolutionary development, ideas regarding the number and design of instruments have undergone considerable change. Instead of the six with which Riggs did his work, sets consisting of a hundred and

fifty or more have been designed and recommended as necessary to successful treatment. In general, these instruments were of two different types and were known as planes and curettes. In general also, the operator using one type of instrument did not use the other: attention was focused on the instrument, rather than on the person using it.

At present all this is largely changed. The pendulum has swung all the way from indifference to enthusiasm and back again to a more common-sense attitude toward the whole subject. Dentists now engaged in periodontia are finding that relatively few instruments are necessary, and that each one must, to a certain extent, make up his own list out of the different sets available. Instead of being divided into two groups, one using curettes and one using planes, many of the leading periodontists of the present are using instruments of both types as they find that they adapt themselves to their requirements. In no other way can one make up a set that will completely meet his needs. What will suit the requirements of one may not prove satisfactory to another.

DIFFERENT TYPES OF INSTRUMENTS

In calling attention, therefore, to some of the different sets of instruments now available, it is not to recommend any one set to the exclusion of another, or to imply that sets of instruments not referred to may not be just as well adapted to the needs of periodontia, but rather to direct attention to instruments that are more or less standard and with which excellent work is being done. Out of these anyone can make up a set adapted to his needs.

THE YOUNGER-GOOD-HUTCHINSON CURETTES

A set of curettes designed by and known as the Younger-Good-Hutchinson set, is one that has stood the test of

time and is well suited to meet the needs of periodontic practice. Most of them are made in "rights and lefts" and so shaped that their blades can be brought to bear upon different angles of the root surfaces of every tooth in the mouth. They have been carefully designed and, when sharp, convey a delicate sense of touch to the fingers of the operator, which tells its own story to those "who have eyes to see and ears to hear." This is an important feature of all instruments used in periodontia, for unless they are delicate enough to convey to the operator the condition of the root surface upon which they are being brought to bear, a full measure of success is impossible.

NEED OF A DELICATE SENSE OF TOUCH

It is equally important that the operator possess a sense of touch which is akin to second-sight, for if this be absent, no matter how well adapted to the work in hand his instrument may be, he will be as one who stumbles in the dark. He may achieve a measure of success, but never success in its fullness. This is not said to discourage the beginner. Anyone recognizing the importance of periodontology and desiring to perfect himself in this branch of dental practice, can do so. While an intuitive sense of what is required is a great advantage, and there can be no doubt but that some possess it, there is no one who cannot acquire a reasonable measure of skill, providing he makes a persevering effort to do so. The treatment of periodontal lesions requires no more skill than that which the dentist is called upon to exercise daily in many other operations in the practice of dentistry. It does, however, require a different kind of skill, one more nearly akin to surgery than to mechanics. A keen sense of touch is of first importance, for the blade of the instrument as it engages the surface of the root is invisible—the only operation in dentistry,

except root-canal therapy, of which this is true. The Younger-Good-Hutchinson set of instruments can be obtained from the Cleveland Dental Manufacturing Company. They are also made to order and most carefully tempered by Charles Grafrath, 556 West 48th Street, New York City.

THE JOHN OPPIE McCALL INSTRUMENTS

A composite set of instruments, thirty in number, including curettes and planes, is one designed by Dr. John Oppie McCall of New York. Most of these are made in "rights and lefts." They differ somewhat in design from the Younger-Good-Hutchinson set, which in some respects the curettes resemble. They are made by the S. S. White Dental Manufacturing Company and consist of eight planes, three sickles, one chisel and eighteen curettes. The author has found numbers 17 and 18 of this set almost indispensable.

THE TOWNER PERIODONTOMES

Another set of instruments, mostly planes, is one designed by Dr. Justin D. Towner of Memphis, Tennessee, and called by him "periodontomes." [1] These are admirably adapted to their purpose. For convenience sake, the root surface of each tooth has been divided into eight different planes and an instrument designed to adapt itself to each of these. They are also made to conform themselves to different angles on differently shaped teeth, as, for example, molars, bicuspids and incisors. And further consideration has been given to whether the teeth to be operated upon are located on the right or left side of the mouth, the upper or lower jaw. The blades are thin, and curved to follow the root surface, and each has two or more cutting edges. The shaft is octagon-shaped, one-quarter inch in diameter,

and hollow. The advantage of this is that it is light in weight in comparison to its size, and affords an extreme sensitiveness to touch. Because of the thinness of the blades, they will enter restricted areas without laceration of the soft tissues. Included in this set are four gum resectors, each of different angles, and an excellent tissue forceps. These are for use in the surgical treatment of advanced cases of periodontoclasia and are splendidly adapted for their purpose.[2] These instruments are made by, and can be obtained of, the Cleveland Dental Manufacturing Company.

THE AUSTIN F. JAMES' PLANES

A set of planes designed by Dr. Austin F. James of Chicago, Illinois, differs from that of Dr. Towner's in that they have a two-point contact upon the tooth which Dr. James believes gives better control of the instrument when in use. As used by him, they yield most satisfactory results. The set consists of 150 instruments and can be obtained of the L. J. Mason Company of Chicago. They are for sale only to those who have taken postgraduate instructions in their use, as their manipulation requires a definite technique.

SHARPENING INSTRUMENTS

It is essential that all instruments used in periodontia be kept sharp, though this is easier said than done. An instrument has been devised for sharpening planes which holds the blade to the stone at the correct angle, thus insuring fairly satisfactory results. Because of the many different angles in curettes, this cannot be done with the same exactness. A rat-tailed-shaped Arkansas stone is often used for this purpose. A carborundum stone, number "186-fine" and made by the Carborundum Company

of Niagara Falls, New York, has been found by the author to be the most satisfactory. This stone, which is four inches long and one inch wide, is wedge-shaped, with the thick and thin edge rounded in such a way as to adapt itself to different-shaped instruments. While it does not meet every requirement it is fairly satisfactory, though no instrument of the curette type can, on the whole, be sharpened as satisfactorily as one could wish, or as well as it is done by the maker. In the use of this stone, the blade is brought to bear on it by laying its concave surface flat on the angle which it most nearly fits and then give it a few strokes back and forth. Care should be exercised to keep the surface *flat* on the stone, for unless this is done one edge or the other will be beveled instead of being ground at an acute right angle to the broad surface of the blade as is desirable. As this takes but a few seconds, one can pause in the midst of the operation to do this, if necessary, and the stone can subsequently be gathered up and included in the sterilizing of the instruments. As their cost is negligible, several may be kept on hand. These instruments also may be returned to the manufacturers for sharpening, if desired.

No matter what type of instrument is used or what method is employed for sharpening it, it is absolutely essentian to the best results that it possess a keen, biting edge. Without this, one will be deceived as to the condition of the root surface that is being operated upon. There is a saying in periodontia that "nothing lies like a dull instrument." Since in the operation of subgingival curettage one has only the sense of touch to guide him, it is absolutely essential that the instrument in use be sharp, for it is impossible to check up on one's work by an ocular examination, as might be done in other branches of dental practice. This is one of the reasons why resort to surgery is so often

made; by it, one is able to visualize the field of operation, which, of course, is a great advantage. Unfortunately, its disadvantages so often outweigh any advantage which may be gained by visibility, that it should be given a subordinate place in periodontia, except in those cases where the only alternative may be tooth extraction.

It will thus be seen that there are many instruments of various designs available to meet the requirements of the most exacting. To learn to use them successfully in the treatment of periodontal diseases requires a long and painstaking experience, especially in the treatment of advanced cases. To undertake treatment, as is sometimes done, with a group of dull, ill-assorted instruments, is to invite defeat. Success in periodontia is not achieved in that way.

REFERENCES

[1] Towner. J. D., 1921, "Standardizing the Technic of Surgical Prophylaxis," *Journal American Dental Association,* vol. 8, p. 225.

[2] Towner, J. D., 1922, "The Surgical Treatment of Periodontal Lesions," *Journal American Dental Association,* vol. 9, p. 706.

CHAPTER XIII

PERIODONTAL ABSCESS

A PERIODONTAL abscess is one occurring in the tissues surrounding the teeth and associated with periodontoclasia and pocket formation. It is frequently referred to as a pyorrheal or parietal abscess. In most cases of pocket formation there is free drainage, in which event there is usually no pain and no inconvenience. But where this does not occur, the surrounding tissues may be invaded by the products of infection and an abscess be formed. What had before been a chronic infection without subjective symptoms becomes an acute one in which there is pain, swelling and inflammation. If allowed to run its course, pus is formed which frequently creates its own fistula somewhere in the involved tissue. With its discharge, the acute symptoms disappear, the fistula usually heals and drainage again takes place through the gingival crevice.

Care must always be exercised not to confuse a periodontal with a periapical abscess, which in many respects it resembles. A differential diagnosis can usually be made by remembering that the latter is always associated with pulpless teeth and occurs within the alveolar process at the apex of the root. Because of their location, the pain connected with periapical abscesses is usually more acute, and the tooth itself more sensitive to percussion.

ETIOLOGY

In the case of a periodontal abscess, the infection is one that takes place in the soft tissues, and while it may include

pulpless teeth, if involved in periodontoclasia, it is more often associated with vital teeth, not because such teeth are more susceptible, but because vital teeth fortunately outnumber pulpless teeth. Since the infection is limited to the soft tissues of the mouth, it is less apt to cause general disturbance, such as rise in temperature, swelling of the face, involvement of the cervical lymph nodes, etc., than is the case in periapical infections. When occurring about vital teeth a differential diagnosis is readily made; when associated with pulpless teeth, this is not so easily done, but is not difficult when all the symptoms are taken into consideration. The most common objective symptom is a localized swelling of the gum about some tooth involved in periodontoclasia. Pain is the usual complaint.

TREATMENT

In the acute stage, treatment is directed toward the reduction of pain and the removal of the cause, which in these cases is some interference with drainage directly from the pocket itself. For this reason, it is necessary that instruments be passed into the pocket through the gingival crevice for the purpose of curetting the root surface and the reëstablishing of drainage. In many cases, the accumulated pus can be discharged in this way. In those cases in which this cannot be done, and pus has already formed in the soft tissues, the abscess should be lanced. If seen before suppuration has taken place, a careful curettement of the periodontal pocket will usually prevent its development. Inasmuch as the soft tissues overlying such pockets are highly sensitive under these conditions, great care should be exercised in the selection and use of instruments so as not to produce trauma; for trauma not only adds to the pain of treatment, but interferes with recovery by spreading the infection. Therefore a careful curettement

of the pocket should be made for the purpose of emptying it of its septic contents at the same time that bleeding is encouraged. Irrigation with a warm, mild antiseptic is important, after which it may be painted with a solution of tincture of aconite and iodine, equal parts.

TREATMENT OF UNDERLYING FACTORS

With the reduction of the acute symptoms, which one treatment will usually accomplish, treatment for periodontoclasia should be begun, for unless this is done the results will be only temporary. It should be remembered that a periodontal abscess is nothing more or less than a periodontal pocket in which a chronic infection has been transformed into an acute one, and that its permanent cure includes the treatment of the preëxisting periodontoclasia. This may require the obliteration of the pocket by surgical means, since, in most of these cases, pocket formation has already reached an advanced stage. This, however, should not be done until after all acute symptoms have disappeared. Under no circumstances should it be allowed to continue as a chronic infection just because the acute symptoms no longer exist. When connected with one of the roots of a pulpless molar tooth, the involved root can sometimes be amputated to advantage. If for any reason the infection cannot be cleared up by treatment, the tooth should be extracted.

PERICEMENTAL ABSCESS

Mention is occasionally made in the literature of dentistry of what is called "pericemental abscesses." [1, 2, 3] In explanation of their etiology it has been affirmed that they are of gouty origin, that somewhere on the surface of the cementum or within the structure of the pericementum, calcium urates in the form of a tophus are deposited.

Others, again, believe that they represent a hematogenous infection and that the primary factor "is usually an accidental trauma or traumatic occlusions." [3] Those entertaining these theories assert that there is no connection between them and the gingival crevice. Roy,[4, 5] on the other hand, believes that this is not so, but that in most instances, possibly in all, they are nothing more nor less than an incorrectly diagnosed periodontal abscess. He cites a case in which he had made a tentative diagnosis of pericemental abscess, when on further examination he discovered that it was simply an obscure type of chronic periodontal abscess with a deep but obscure pocket on the lingual surface which was discharging through a sinus on the labial surface. His conclusions are as follows: "I am convinced that if the practitioner be willing to institute a most minute investigation in all cases of so-called pericemental abscess about vital teeth, he will find what I have found, and will acknowledge in principle that pericemental abscesses upon vital teeth are pyorrhetic abscesses formed in a serpiginous pocket whose cervical opening is not recognized because of its location at a point some distance from the gingival abscess." [4] As the author has never seen a "pericemental abscess" he is inclined to agree with Roy, that probably that which has been diagnosed as such was nothing more nor less than a periodontal abscess which was incorrectly diagnosed, for reasons pointed out by Roy. When a case presents itself in which there is an abscess in the periodontal tissues surrounding a vital tooth, a most searching examination should be made to ascertain whether there is not a pocket somewhere on the circumference of the involved tooth. If such prove to be the case, the diagnosis is clear. It should be remembered that in the earlier days when the diagnosis of pericemental abscess was first made, the radiograph was not in common use, and knowledge

regarding periodontal infections had not progressed to the point it now has.

REFERENCES

[1] Kirk, E. C., 1898, "Abscesses Upon Teeth With Vital Pulps," *Dental Cosmos,* vol. 40, p. 621.

[2] Kirk, E. C., 1913, "The *Locus Minoris Resistentiæ* in Pyorrhea Alveolaris," *Dental Cosmos,* vol. 55, p. 577.

[3] McCall, J. O., 1918, "Primary Factors in the Etiology of Periodontoclasia," *Dental Cosmos,* vol. 69, p. 1091.

[4] Roy, M., 1918, "The Nature of Pericemental Abscesses upon Vital Teeth," *Dental Cosmos,* vol. 60, p. 573.

[5] Roy, M., 1918, "The Pathogenesis and Prophylaxis of Pyorrhea Alveolaris," *Dental Cosmos,* vol. 60, p. 668.

CHAPTER XIV

DIET

The important part which diet plays in health and disease makes it necessary to consider it in its possible relationship to diseases of the periodontium, especially periodontoclasia. Much has been said and written on the subject in recent years, and much has been done in the way of animal experimentation to determine whether or not any such relationship exists. That certain changes occur in the osseous support of the teeth as a direct result of alterations in diet has been amply proved by many investigators, notably by Howe, Grieves, Jones and Simonton. These changes are represented by absorption of the alveolar process which, in many respects, resembles the diffuse atrophy of Gottlieb. Whether this resorption is of the same nature as that which takes place in periodontoclasia, is far from certain. Gottlieb [4] is of the opinion that, in many instances, the resorption of the alveolar process shown in animal experimentation is an entirely different process from that seen in cases of human periodontoclasia, and that the phenomenon of bone resorption seen in these cases is not a part of the syndrome of periodontoclasia, but is in reality an actual scurvy. That there is a type of periodontoclasia in which errors in diet are an important etiologic factor is the opinion of Simonton.[5] He calls attention to certain tissue changes occurring in a 3½-year-old dog which had been subjected for 130 days to a diet in which the alkaline ions were in excess of normal. These changes were represented by "resorption of the alve-

olar septum, lateral excavations, opening the bone marrow spaces to the side, osteoporosis, coalescing of the marrow spaces and widening of the sockets of the teeth crownwise." These abnormalities he believes to be comparable to those taking place in certain cases of human periodontoclasia, and which are often seen in radiographic examinations. It is this type that is referred to by him as *periodontoclasia obscura.*

From the foregoing it must be evident that the health and integrity of the alveolar process may be profoundly influenced by alterations in diet as shown by animal experimentation, and that in some cases, at least, abnormalities in diet must be reckoned with as one of the possible etiologic factors in human periodontoclasia. For these reasons, its consideration is a matter of importance, though it must be admitted that the part which it plays in the breaking down of the alveolar process and the way in which it operates to do this are matters about which there is, at present, no exact knowledge. The difficulty in every case is to determine how much of the pathologic syndrome is due to disturbance in nutrition, and how much to purely local conditions. Unfortunately, there is no set rule to guide one in evaluating the different etiologic factors which may be present in a given case, nor what steps it may be necessary to take to remove them. In a general way it may be said that where the bone resorption is marked, involving more or less all of the teeth, and in which the local factors do not seem at all commensurate with the destruction, one should always suspect disturbances in nutrition, especially in young patients.

VITAMINS

All foods contain a vital element called vitamins. These are five in number, A, B, C, D, and E.[9] They are all more

or less injured and their nutritional value limited by such factors as heat, storage, canning, drying and by various manufacturing and refining processes. The further removed any food is from its natural state, the greater will be its vitamin deficiency.[2] Foods cooked by steam are injured less than by any other method of cooking. It is only rarely that human foods are totally devoid of any one vitamin. Their deficiency is more often partial than complete, being associated most frequently with a deficiency of proteins and inorganic salts. A diet in which any one of them is long deficient leads to all manner of disturbances such as loss of appetite, indigestion, constipation, diarrhea, anemia, weakness, nervousness, neuralgic pains, etc. They represent the life-giving element in all foods. One could gorge himself to repletion, but if the food had been partially robbed of its vitamin content, the individual could be literally starved because of vitamin deficiency. The preparation of most foods tends to injure their vitamin content, which, coupled with a limited dietary so often the result of habit, explains many cases of nutritional unbalance, with its sequel of ill health and disease.

VITAMIN A

Vitamin A is found most abundantly in green uncooked leaves such as lettuce, cabbage, spinach, alfalfa, clover, green grass, etc.; in certain fruits; also in butter fat, egg yolk, cod-liver oil; in all glandular tissue such as kidney, sweetbreads, liver, etc. Of these latter, liver is of especial value because of its high content of all vitamins. Foods in which vitamin A is deficient or absent altogether are vegetable oils, skim milk, white flour, pure corn flour, polished rice, cane and beet sugar, syrups, meat extracts, proprietary and highly refined foods, and all white fish. Considerable vitamin A is stored in the body fats. This is

highly important in young and growing animals and in adults for the maintenance of health and the powers of reproduction. The ultraviolet rays of sunshine are highly important in the production of vitamin A in the body fats and in the overcoming of excessive acidity. When taken in normal quantities vitamin A conduces to vigor of body and a youthful appearance.

VITAMIN B

The chief sources of vitamin B are whole grains, peas, beans, lentils, fruits, nuts, green vegetables, yeast, asparagus, heart muscle, sweetbreads, liver, kidney, and fish roe. It is absent from white flour, corn flour, polished rice, tapioca, egg substitutes, cod-liver oil, butter, cream, meat extracts, olive oil, sugar, tea and coffee, and deficient or wholly lacking in many of the refined foods of modern civilization. It is slowly destroyed by heat, especially so in the process of baking. It is dissolved out by water which makes it desirable that, as far as possible, all vegetables be cooked by steam. At no time does the body have more than a small reserve of vitamin B. When absent from the diet, susceptibility to infection and neuritis is greatly increased, and bodily growth stunted.

VITAMIN C

Of all the vitamins, vitamin C is the most unstable and easily destroyed. Heating it to 110 degrees Fahrenheit is sufficient to destroy it in many instances. Age and storage also affect it adversely. Green leafy vegetables and raw ripe fruits are the foods richest in vitamin C, but it is desirable that they be eaten soon after picking because of the unstable characteristic of this vitamin. It should also be remembered that all the vitamins in fruit, vegetables, etc., are found in greatest abundance in the protective outer

DISTRIBUTION OF THE VITAMINS

Nomenclature

* Means small amount. *** Means large amount.
** Means fair amount. **** Means exceptional amount.

Source	*Vit. A*	*Vit. B*	*Vit. C*	*Vit. D*
Apples	*	**	**	
Asparagus		***		
Beans	*	***		
Beets		*	*	
Bread (white)		*		
Bread (whole W.)	**	**		*
Butter	*to***			*
Cabbage (raw)	**	**	***	
Carrots (raw)	**	*	**	
Celery (green)	**	**	**	
Cheese (cream)	**	**		*
Cod liver oil	***			****
Cream (Summer)	***		*	*
Eggs (Summer)	***	*		*
Lard				
Lemons		**	***	
Lettuce	**	**	***	
Milk (raw, Summer)	***	**	**	**
Milk (Evap.)	***	**		*
Olive oil				
Orange juice		**	***	
Peaches	**		**	
Pineapple	**	**	***	
Potato (white)	*	**	**	
Rice (whole)	*	**		
Spinach	***	***	***	
Sugar				
Tomato	**	***	***	
Turnip		**	***	
Wheat (whole)	**	***		
Yeast		***		

Vitamin A protects tissue of head, thorax, sex glands, etc.
Vitamin B is anti-neuritic and growth-promoting.
Vitamin C protects tissues of the mouth; favors bone formation.
Vitamin D is growth-promoting and antirachitic.

FIG. 51.—Chart showing distribution of vitamins. Arranged by Dr. Sherman L. Davis of the Indiana University School of Dentistry.

coat or peelings. Vitamin C is found in summer milk and cream, but is absent in butter and cheese, sugar, whole wheat, rice, brans, cod-liver oil, etc. Absence from the diet of vitamin C causes scurvy, inflamed and bleeding gums, hemorrhage, etc., and interferes with the proper clotting of the blood and the metabolism of mineral salts.

VITAMIN D

Cod-liver oil is the richest source of vitamin D. Dairy products and eggs contain a moderate amount during the summer, when cows and hens are exposed to the ultraviolet rays of the sun. It is usually absent altogether in these foods during the winter. Of all the vitamins, it is the least widely distributed and therefore most apt to be lacking in the dietary. It is an important factor in promoting the growth of bone, cartilage, ligaments and muscles, and should always be incorporated in the dietary of pregnant women. In some way the ultraviolet ray seems to increase its efficiency. For this reason cod-liver oil is frequently activated by exposing it to its influence.

VITAMIN E

This is a newly discovered vitamin believed to be present in most natural foods and one that is not easily destroyed by cooking or the addition of baking soda. All other vitamins are quickly destroyed by baking soda and other alkalies used in the preparation of foods. The absence of vitamin E from the diet is believed to cause sterility.

DIET DEFICIENCY

Errors in diet, as they affect the health of the individual, usually take the form of diet deficiency; that is, the diet is not well rounded, being deficient in certain nutritive elements. It may also take the form of disturbance in the

acid-base balance as shown by Simonton in his experiments on dogs.[8] It is impossible to maintain, in health, any of the tissues of the body if they are being deprived of food elements essential to their nutrition and growth. Unfortunately, this is often done through ignorance in the selection of foods. As is well known, habit often governs in this important matter, foods being chosen without any regard whatever for the needs of nutrition. In many instances, also, the modern preparation of foods robs them of much of their nutritive value, as does prolonged and excessive heat in cooking. The result is that people may starve themselves in the midst of plenty, so far as certain food elements are concerned, and do so without the slightest knowledge of that fact. Any diet that injures the periodontal tissues by robbing them of their nutritional needs will also injure the general health, though this latter may be less obvious. At the same time, a diet that promotes a high standard of bodily nutrition is of great value in providing the periodontal tissues with the nutritive elements essential to their health and well-being. Such a diet is generally comprehended in the term, "balanced diet."

BALANCED DIET

According to McCollum and others a "system which can be recommended with assurances that it will go a long way toward improving the physical fitness of the nation" [5] is:

First: The extension of the use of dairy products. This is most important. It should include the use of not less than one quart of milk per day. Through its encouragement of the growth of lactic acid-producing organisms, milk serves to bring about the disappearance from the intestinal tract of those bacteria causing putrefactive decomposition. It has, moreover, dietary properties which make it the one food for which there is no effective substitute. It is said

to supplement any diet to which it is added in respect to the quality of its proteins, its organic content, and the presence in it of the three dietary essentials, vitamins A, B, and C. During the summer, when cows are in pasture, it also contains vitamin D. It is rich in its calcium content, which makes it a valuable food in cases of dental caries and periodontoclasia. Milk is deficient only in iron. The other dairy products, such as cream, butter and cheese, are also of great value, and should form a part of the diet.

Second: Leafy vegetables. These are unique among foods of vegetable origin. They include lettuce, spinach, cabbage, brussels sprouts, celery, chard, beet leaves, endive, watercress, etc. Their consumption in liberal quantities, as regular constituents of the diet, not only serve to provide the body with valuable nutriments it cannot secure in adequate amounts in any other way, but also serves to maintain the intestinal tract in an hygienic condition by promoting prompt elimination. They are rich in iron and calcium, both of which are important dietary elements.

Third: Vegetables. The diet should also include the taking regularly of a certain amount of vegetables, raw and cooked, for the purpose of supplying the body with a sufficient quantity of antiscorbutic substance. Such vegetables as radishes, turnips, carrots and onions can be eaten raw and could to advantage be made a part of the regular diet. They provide the body with the important mineral elements of potash, soda, lime, iron sulphur, magnesia, etc. All vegetables, cooked or uncooked, should form a considerable part of the diet of every individual, though no vegetarian diet can ever be entirely adequate to the needs of nutrition.

Fourth: Fruits. In the selection of foods, fruits must be given an important place. To a larger degree than any other food, they are conducive to health and longevity.[1]

They maintain in normal condition the alkalinity of the blood, while more concentrated foods such as cereals, pulses, cheese, meat, etc., are more or less acid-forming. The alkaline elements which are chemically combined with fruit acids act as a natural laxative by promoting the action of the secretory glands. They supply the elements of potassium, calcium, iron and phosphorus in a highly organized form, and in this respect are far superior to cereals. Many of them, including berries, are especially rich in organic iron compounds, so important in replenishing the red-blood corpuscles. They are also excellent sources of vitamins B and C, the latter being found chiefly in citrous fruits, which makes them a valuable article of diet and one of the best remedies for anemic conditions. While fresh fruits do not possess the high nutritive value of the more concentrated cereals, legumes and nuts, they are, nevertheless, indispensable in the maintenance of health and vitality. They all contain much water and varying amounts of sugar, but are relatively insignificant as sources of proteins or fats. Kidney, liver and milk stand out as a group of foods containing proteins of unusual value. Among cereal grains, whole wheat easily stands first in the quality of its proteins.

The best type of diet is undoubtedly one which, in addition to moderate amounts of cereals, tubers, legumes, root vegetables and small amounts of meats and eggs, contains liberal quantities of milk, fruits and leafy vegetables. It is the use of these foods in proper proportions that constitute a so-called balanced diet, and one which can in most cases be followed to advantage.[5]

In the treatment of periodontoclasia, the dentist should make inquiry regarding the dietary habits of his patients, and take such steps as may seem necessary to correct them. It should always be borne in mind, however, that nutrition is not only a matter of foods, but one of assimilation also,

and that individuals differ widely in their assimilative capacity. While one can hardly make a mistake in recommending in the dietary such foods as dairy products, leafy vegetables, raw fruits, etc., it must not be forgotten that there are cases in which other factors have to be considered, and that generally the patient's family physician is the one best qualified to prescribe for these cases.

REFERENCES

[1] Carque, Otto, 1923, "Rational Diet," *Times-Mirror Press*, Los Angeles.

[2] Fones, A. C., 1923, "Diet in Relation to Teeth," *Dental Cosmos*, vol. 65, p. 519.

[3] Froude, C. C., 1923, "Right Food," Brentano's, New York City.

[4] Gottleib, B., 1929, "Paradental Pyorrhea and Alveolar Atrophy," *Dental Digest*, vol. 35, p. 244.

[5] McCollum, E. V., 1922, "The Newer Knowledge of Nutrition," The Macmillan Company, New York City.

[6] McCollum, E. V., and Simonds, Nina, 1920, "The American Home Diet," F. C. Mathews Company, Detroit, Mich.

[7] Orban, B., 1927, "Nutrition and Teeth," *Journal American Dental Association*, vol. 14, p. 1619.

[8] Simonton, F. V., 1927, "The Etiology of Paradontochasia," *Journal American Dental Association*, vol. 14, p. 1780.

[9] Donnelly, L. C., 1929, "Food and Light." Reprint.

CHAPTER XV

POSTOPERATIVE CARE. THE DENTIST'S PART

It will be remembered that in considering the treatment of periodontoclasia, the subject was divided into two parts; one having to do with operative, and one with postoperative procedures, and that the latter had for its object the control of those etiologic factors which are more or less common to all cases, to the end that health in the periodontal tissues be reëstablished and maintained. These factors, it will be recalled, were bacteria and low-cell resistance. Their control is absolutely essential in the postoperative care of all diseases involving the periodontal tissues. Indeed, all postoperative efforts of whatever nature have for their object the reduction of bacteria through the application of the principles of mouth hygiene, and, what is equally important though less generally recognized, the stimulation of periodontal circulation with a view to increasing nutrition and raising resistance to infection in the tissues which surround and support the teeth. The ways and means by which this is achieved, the part which each must play—dentist and patient—in its successful accomplishment, is what is meant by "postoperative care." Too much in the past has reliance been placed on the operative efforts of the periodontist, and too little on the postoperative care of the patient. The first has to do with the removal of all those factors which have developed in the periodontal tissues as a result of disease and without which no progress in treatment can be made; the latter represents the effort which must be made to arouse those biologic forces resident

in the tissues themselves, in order that the fruits of operative treatment be established and maintained. One is as important to complete success as is the other, though naturally the operative treatment must in all cases precede and prepare the way for the important part which both the dentist and patient must play in the postoperative care of each and every case.

As already indicated, the postoperative care of all cases of periodontal disease involves a dual responsibility, in which the dentist plays the leading rôle, though that played by the patient is no less important.

THE DENTIST'S PART

The dentist's part in the postoperative care of the patient consists in (*a*) carefully instructing the patient in correct methods of mouth toilet; (*b*) placing in his hands proper instruments with which to accomplish this, such as toothbrushes, dentifrices, waxed silk tape, toothpicks, etc.; and (*c*) such supervisory care in the way of regular prophylactic treatments, repeated instructions, etc., as may be necessary to maintain the patient's interest and the periodontal tissues in health. Until these things have been done by the dentist, the patient cannot be expected to perform his part intelligently, nor be held responsible for the outcome. The sympathetic coöperation of both is essential, if good results are to be expected. In a sense, this is unfortunate, as neither can wholly succeed without the coöperation of the other. For this reason, great care must be exercised in obtaining this coöperation.

(A) INSTRUCTIONS TO PATIENT

Instructions as to the patient's home care of the mouth should begin with the first visit, and be continued until the skillful care of the mouth has become a fixed habit. In

order that the patient may perform his part intelligently, the important rôle which bacteria and low-tissue tone play in the etiology and progress of all periodontal diseases should be explained to him in plain and simple language. If he be told to brush his teeth and massage his gums, without any explanation of why he should do these, he is less apt to persevere in well-doing as the immediate need for such care becomes less obvious. As the symptoms of disease disappear, as they will do, it is only natural that the patient should relax in his care, unless in the first instance he has been made to understand why his day-by-day care is essential to *continued* success. These verbal instructions should be supplemented by clinical demonstrations of the means by which these very important objectives are achieved. This may be begun by a demonstration in one's own mouth of the correct use of the toothbrush in its dual capacity of tooth cleanser and gum stimulator. The purpose of both should again be emphasized and the fact reiterated, that one is as important as the other. This should be followed later by a toothbrush demonstration in the patient's mouth, while he observes the operator in a hand mirror. He is then given the brush, and asked to try his hand at it and his efforts criticized in a constructive, and as far as possible, encouraging way. One should be careful not to antagonize or discourage the patient as may be done if one is too severe in his criticisms. "Men are but children of a larger growth" and are more easily led than driven. In many cases, indeed in most, it will be necessary to take the patient's hand in one's own, and guide the movements of the toothbrush in its different excursions in the mouth.

No attempt should be made to teach the whole lesson at one visit. After the first demonstration, request the patient to follow instructions as carefully as he can and to

observe for himself any difficulties in technique which he may encounter, and to report them at the next visit. Further instructions and demonstrations are then given, and should be continued until the lesson has been fairly learned. As in every other endeavor to impart information, it will be found that there are those who learn readily and those who do not; those who persevere in well-doing and those who fall by the way, and unfortunately there are those who never profit by their instruction, and who are satisfied to continue in their old "hit or miss" habits. It is these latter cases that try one's soul. Unfortunately this type of patient, "like the poor, is always with us" and must be reckoned with. They are the thorns, so to speak, in one's professional side, the type of patient that inculcates, by repeated derelictions, the lessons of patience. Such patients should not be dismissed from one's practice as one is sometimes tempted to do, and as has been recommended, for though they do not achieve a full measure of success, something at least can and should be done for them. In these cases one should examine himself to see whether or not the fault may not be that of the teacher. This undoubtedly explains some failures, though by no means all of them; some patients are habitually careless. Having exhausted all one's resources in this direction, the patient should be tactfully told of his neglect of duty, and that he must assume responsibility for the results. This with the date should be made a part of the record, for patients sometimes have a way of forgetting when things do not turn out well. Under such circumstances it is a satisfaction to be able to turn to the written record and remind the patient that on such and such a date he was warned of what might be expected if he persisted in his indifference. This, of course, should not be done except

when faced by a complaining patient and never in the spirit of "I told you so."

(B) BRUSHES, DENTIFRICES, WAXED SILK TAPE, ETC.

It is also the dentist's responsibility in the postoperative care of his patients to see to it that they are equipped with the proper tools with which to carry out his instructions. Because the patient brushes his teeth two or three times daily, it is not safe to assume that he has either the proper brushes or dentifrices for his needs. If one is in doubt on this point, let him ask a dozen of his patients to bring in the brushes they are using and observe for himself what they look like. The probabilities are he will not find one that measures up to the patient's needs. Instead, he will find a dozen ill-assorted, unclean, badly worn and poorly shaped brushes, which it is impossible for anyone to use with any degree of success. So far as the patient's requirements are concerned, the same will be true of dentifrices. His first duty, therefore, is to study his patients' needs regarding toothbrushes, dentifrices, etc., and see that they are provided with such as meet their requirements. In order to do this intelligently, he should inform himself on the qualifications of both, and having done so, make arrangements with his druggist to keep them in stock.

As might be expected, there is some diversity of opinion as to what constitutes a good brush. In the days preceding the preaching of the gospel of oral hygiene, and to some extent even down to the present, the value of a toothbrush was generally measured in terms of size; hence large brushes were the style. The maker was not altogether at fault in this, for he gave his patrons what they asked for, and they, with no one to instruct them in what they should use, purchased those that gave them the most for their money.

In more recent times, however, the pendulum, as might have been expected, has been swinging in the other direction. Brushes with one or two rows of bristles and less than an inch in length are being offered to the public and being prescribed by the dentist for adult use. There should, of course, be toothbrushes of different sizes to meet individual needs and they need not all be of the same design; nevertheless, toothbrushes can be too small, just as in the past many of them have been too large. This may sound like heterodoxy, but if one will lay aside any prejudices which he may have, and will study the toothbrush in action, he must be convinced that brushes are being made and recommended that are too small to measure up to present-day conceptions of oral hygiene or the demands which are being made upon patients in the personal care of their mouths. This is not to say that we should revert to large toothbrushes, but to encourage the recommendation of those of such size, quality and design as will, when in use, give the maximum of results with the minimum of effort. This cannot be done with "freak brushes."

For general all round purposes it may be said that an adult's brush should be from one and one-half to one and three-quarter inches in length; three-eighths to one-quarter of an inch wide at the heel, tapering slightly toward the point, with bristles varying from three-eighths to one-half of an inch in length. If made longer than this, as is frequently the case, they interfere with the free movement of the brush in the mouth, at the same time that they tend to buckle on themselves and break off when in use. Moreover, the cleansing and stimulating effect of a brush is seriously impaired by bristles that are too long. This will be self-evident to anyone who will make a comparative test, in the mouth, of brushes with long and short bristles.

These should be three rows wide and each tuft should be made up of bristles of different lengths, those in the center being longest. In this way, each tuft or row of tufts terminates in a point. These tufts should be separated from each other sufficiently to permit of flexibility, but not enough to create wide spaces between each row, as such brushes are not as efficient in action nor as easily kept clean. The handle, not including the brush itself, should be four and one-half to five inches in length, and should be bent slightly backward. When bent forward, as is sometimes the case, the brush is awkward to handle and much less efficient in action. It should be rigid enough to prevent the slightest flexibility when in use. For this reason brushes with composition handles are, as a rule, less satisfactory than those with bone handles. All edges should be beveled, so as not to interfere with the free use of the brush in the mouth. Brushes should also be numbered to insure identification when several members of a family are using the same design.

Not infrequently it is desirable to prescribe brushes of slightly varying designs and sizes to meet individual needs and to be used in rotation. In this way, different surfaces of the teeth are reached that might not be if one were always to use a brush of a given design. For these reasons it may be wise in most instances to provide patients with at least two different brushes of unlike designs. Sometimes the inclusion of a "freak" brush in the collection of those in daily use may work out well in that it may reach certain areas that the more orthodox brush designed for general use fails to do. Each case should be studied by itself and brushes prescribed to meet the needs of that particular case, always bearing in mind that it is unwise to complicate the operation of toothbrushing by intricate instructions or a complexity of toothbrushes.

HARD AND SOFT BRUSHES

"Hard," "medium" and "soft," as applied to the texture of toothbrush bristles, are relative terms which mean different things to different makers. One should therefore inform himself regarding their meaning as applied to the brushes which he may be recommending, in order that he may prescribe them intelligently. The bristles should be flexible without being soft, and stiff without being coarse. Brushes can be too hard and too soft. The brush with the coarse unbleached bristles usually belongs to the first group, and those with hair bristles belong to the second. Both represent extremes and, generally speaking, have a limited place in the daily care of the mouth.

In those cases in which the gums are unaccustomed to the stimulating influence of the toothbrush, it is usually well to begin with a brush of soft or medium texture, and gradually work up to a harder one. As a rule, a brush that is reasonably hard should be used, and when properly employed is of great assistance in reducing bacterial accumulations and in promoting periodontal health by stimulating the circulation.

FREQUENCY WITH WHICH TEETH SHOULD BE BRUSHED

The frequency with which teeth should be brushed will naturally vary with varying conditions. No hard-and-fast rule can be laid down. One brushing operation, intelligently performed, is better than a half dozen indifferently done. Efficiency, rather than numbers, should be emphasized, though both are important. An excellent rule to hold up to the patient and one which has the virtue of being logical, is to brush the teeth before breakfast and after each meal.[2] The first brushing operation should be before breakfast and made a part of the morning toilet.

During the night, when there is nothing to disturb their growth, the bacteria of the mouth, as stated by Hartzell, double their number every thirty-five minutes.[10] Furthermore, a certain amount of stagnation of the oral secretions occurs. For these and other reasons, it is highly desirable that the teeth be brushed before breakfast. This should include a careful brushing of the gums and tongue also. If preferred, instead of brushing the tongue a tongue-scraper can be used and is probably more efficient. The chief purpose of this first brushing operation is to cleanse the mouth of bacterial accumulation, remove mucin plaques, polish the teeth and stimulate periodontal circulation.

The after-breakfast and after-lunch brushings have for their purpose another object; namely, the removal of food débris from around and between the teeth. It is, so to speak, an operation for the sweeping up of the food litter. In those cases which have been under treatment for periodontoclasia, a little time can also be profitably devoted to gum stimulation. When abnormal contact points are present, or where the gums are receded, creating thereby triangular-shaped spaces between the teeth, waxed silk floss should be used. This should in all cases precede the brushing operation. The loosened particles of foods can then be washed out, as far as that is possible, by thoroughly flushing the mouth with a solution of salt and water, one-quarter teaspoonful of salt to a half glass of warm water, or in those cases where the exposed root surfaces are sensitive, lime-water can be substituted for the normal salt solution. In healthy mouths, plain warm water will serve the purpose, as the object aimed at is chiefly that of washing out of the mouth the loosened food particles. When this has been done, the brush is again used to remove any food débris which may have eluded the use of silk and

mouth wash. A dentifrice is unnecessary in either of these brushings, for, as already indicated, these are not primarily tooth-polishing but food-sweeping operations. Neither need consume much time.

The fourth daily brushing should be done immediately after dinner at night. This is better than to wait until bedtime, for, in that event, fermentation of food particles is more likely to occur. This brushing serves the double purpose of removing food particles and polishing the teeth, plus a final stimulation of the gum tissues. Therefore, the teeth should be silked, the mouth rinsed, the teeth polished and the gums stimulated. A dentifrice should be used and the teeth given one of the most thorough brushings of the day. Unless food is taken before retiring, they need not again be brushed. The technique of toothbrushing will be outlined later.

NUMBER OF TOOTHBRUSHES USED DAILY

The patient should be instructed not to use the same brush twice in the same day. In this way, the brush is given an opportunity to dry out and is always fresh and hard, and therefore more efficient. As already indicated, these brushes may be of different designs to meet the varying conditions met with in the mouth. They should, however, all have bristles of fair stiffness, and should be discarded as soon as they become soft and replaced with new ones. This cannot be made too plain. The thing to impress upon the patient is, that in the final analysis it is not so much a matter of *toothbrush* as of *toothbrushing,* and that in most instances it is the patient and not the brush that explains the difference between a good and a poor operation. It is usually a poor workman who blames his tools.

CARE OF THE TOOTHBRUSH

The proper care of the toothbrush is a matter of importance. All are agreed that it should be kept clean. Some believe that it should be sterilized after using, though no one has devised a wholly satisfactory way in which to do this. If this could be done in a simple and efficient manner and without injury to the toothbrush, it, no doubt, would be desirable, since a brush that was both clean and sterile would in these respects be above criticism. At present, this is not possible and dependence must therefore be placed upon cleanliness. And in most instances, except in cases of Vincent's infection, this is all that is necessary. The mouth itself is not sterile and cannot be made so. Neither is much of the food that is eaten sterile, nor the dishes upon which it is served, nor the utensils with which it is placed in the mouth. It is important that these be clean, but no one insists that they be surgically sterile. In the same way, the toothbrush should be kept clean and, under such conditions, may be used with entire safety. It is, of course, assumed that one uses his own, and not his neighbor's, toothbrush.

After having used the brush, a satisfactory way in which to care for it is to hold it under the faucet and allow cold water to forcibly flow over it. Hot water softens the bristles without sterilizing them, and sometimes distorts the shape of the handle. Then, before putting it in an upright position to dry, and after having shaken out the excess of water, it is liberally sprinkled with salt, which is permitted to remain on it until it is used the following day. The excess of salt may then be washed off and it is again ready for use. A simple way in which to accomplish this is to instruct the patient to fill an ordinary tooth-powder bottle with table salt and sprinkle it on the brush, after the completion of the brushing operation, just as is done in the

use of a tooth powder. While the salt, as generally used, may not completely sterilize the brush, it will restrain bacterial growth and do all that is necessary to keep the brush in a satisfactory condition. Moreover, enough salt will remain on the brush to exercise a stimulating effect upon the tissues of the periodontium.

The therapeutic value of salt in the prevention and treatment of periodontal diseases has never been fully appreciated.[9] Finely powdered and used as a dentifrice in cases of soft and bleeding gums, it is most helpful. The addition to it of a small quantity of light calcined magnesia not only reduces somewhat the strength of the salt, which is desirable, but also prevents it from absorbing moisture and so becoming lumpy. It can be flavored in such a way as to make its use very pleasant. Following subgingival curettage, the exposed surfaces of the teeth are sometimes sensitive, which may make the use of a sodium-chloride dentifrice inadvisable immediately after operating. This is usually only temporary. An excellent formula which can be put up by any druggist is the following:

Sodium chloride (powdered)	14 drams
Magnesia calcined (light)	2 drams
Oil of wintergreen	45 minims
Oil of peppermint	10 minims

Sig: Use as a tooth powder two or three times daily.

Both flavoring agents may be omitted, if desired, and the strength of the salt reduced by changing the relative amounts of the sodium chloride and calcined magnesia. This latter may be desirable in some cases.

THE USE OF DENTIFRICES

In cleansing the mouth and teeth a dentifrice, properly compounded, is useful, just as a cake of soap is helpful in

washing the hands. As a rule dentifrices have little or no therapeutic value, and in the healthy mouth none is needed or desired.[6] Their function is to assist in the cleansing operation and to do this without injury to either the teeth or gums. In cases of disease, dentifrices may be compounded that, theoretically at least, possess some therapeutic influence and may be used to advantage; as, for example, a dentifrice containing sodium perborate for use in the postoperative treatment of Vincent's infection, and sodium ricinoleate in cases of periodontoclasia where it is desirable to supplement the patient's efforts at toothbrushing by the use of a germicidal dentifrice. Their chief value consists in their abrasive and detergent effects. They may, however, be too abrasive, and so cause injury to the teeth, as is often done. For these reasons it is important that one prescribe dentifrices the exact composition of which is known. To save the trouble of writing a prescription at each time one may be needed, those that are used frequently may be left with one's druggist from whom the patient is directed to obtain it. These can be designated by some symbol known to both dentist and druggist. In case of tooth pastes, where it is necessary that they be put up in fairly large quantities to obtain the best results, this is especially desirable. Moreover, it has the added advantage of being available when called for, thus making it unnecessary for the patient to wait while the prescription is being filled. In most places the druggist maintains a delivery system, in which case the prescription may be sent direct to him with instructions regarding delivery. Postal cards used as a prescription blank can be used in such cases. These can be addressed in advance of one's needs, and so be ready for use at any time. Where this is inconvenient, the prescription should be given to the patient who can

have it compounded anywhere. A careful record of all prescriptions should be kept as a part of the patient's history chart.

The following are prescriptions for dentifrices that can be put up by any druggist and will meet practically every need of the ordinary case. They can be modified to meet almost any situation which may arise in daily practice. For example the sodium perborate in a dentifrice can be increased or decreased as may seem best suited to the case in hand. The same is true of the sodium ricinoleate, which can be omitted altogether if desired. Some patients have an aversion to certain flavoring ingredients. This, again, can be arranged to suit individual preferences. It should always be remembered that it is difficult to put up tooth pastes in small quantities. For this reason it is desirable that arrangements be made with one's druggist to put these up in such quantities as may be necessary to obtain the best results, and refer the patient to him when prescribing them. All dentifrices should be of simple construction, their primary purpose being to assist in the cleansing of the hard and soft tissues of the mouth without injury to either. It is unwise to attempt "shotgun" formulas for use in dentifrices.

Tooth powder for daily use:

Calcium carbonate precip.	8 drams
Sodium perborate	4 drams
Powdered castile soap U. S. P.	40 grains
Saccharin	1½ grain
Oil of wintergreen	15 drops
Oil of peppermint	2 drops

Sig: Use on toothbrush night and morning in brushing teeth.

The following powder is one that is distinctly alkaline in reaction and may be prescribed in cases of acid mouth, so-called:

Calcium carbonate precip. ..... 5 drams
Magnesium carbonate ..... 5 drams
Powdered castile soap U. S. P. .... 30 grains
Oil of peppermint ..... 10 drops
Sig: To be used night and morning in brushing teeth.

The following tooth paste can be put up by any druggist, but when put up in small quantity, as for example a single tube, it is not always satisfactory:

Calcium carbonate precip. ..... 2 ounces
Sodium perborate ..... ½ ounce
Powdered castile soap U. S. P. .... 2 drams
Sodium ricinoleate 40% ..... 1 dram
Glycerin
Oil of wintergreen
Oil of peppermint ..... aa qs.ft. tooth paste
Sig: Use on toothbrush two or three times daily in cleansing teeth.

The reader who is interested in the study of dentifrices in their application to dental practice is referred to the following authors: Buckley, *Dental Cosmos,* vol. 52, p. 429; Kelly, *Journal Allied Dental Society,* vol. 8, p. 144; McClelland, *Dental Cosmos,* vol. 66, pp. 425, 751, and McGehee, *Journal American Dental Association,* vol. 13, p. 1599.

THE USE OF MOUTH WASHES

The regular and persistent use of mouth washes is usually unwise.[5] As sterilizing agents they are not only ineffective, but their continued use may prove harmful. In some instances it is believed that they act as salivary depressants by diminishing the flow of saliva at the same time that they promote stagnation. Many of them contain astringents which tend to inhibit the blood supply in tissues of the periodontium and so act to defeat the most important principle in promoting health in these tissues. People have got the mouth-wash habit and are often disappointed when

the dentist does not prescribe some miraculous mouth wash that is going to cure them of all the dental ills to which the flesh is heir. This is unfortunate and needs to be corrected. In certain diseases of the mouth, washes have their place, and they should be prescribed, but only to be used as a part of the treatment, and to be discontinued when the condition for which they were prescribed ceases. As in the case of dentifrices, it is desirable to write one's own prescriptions, as in no other way can one be sure of what is being used. The daily use of medicated mouth washes as a part of the toilet should be discouraged.

In cases of active caries, a saturated solution of lime-water has stood the test of time, and is generally regarded as helpful as a preventive measure. Dr. Alfred C. Fones in his book on "Mouth Hygiene," page 291, recommends that the patient make his own solution by obtaining coarse lime at a paint store or of a mason, such as is used in making rough plaster. This should be cream-white in color. The lumps are broken up into a coarse powder and half a cup full put into a quart bottle or jar and nearly filled with cold water. This should be vigorously shaken and allowed to settle for three or four hours. The container should not be tightly stoppered. The liquid should then be carefully drawn off and the receptacle refilled and again shaken. Five or six quarts can be made in this way and at very little expense. If it seems a little strong at first, it should be diluted. As it is not altogether pleasant to the taste, the patient should be advised to wash the mouth with warm water after using. This wash is also helpful in those cases of periodontal lesions where, as a result of gingival recession, the necks of the teeth are sensitive to touch. As this may be caused by acids, the neutralizing effect of the lime-water will be beneficial in such cases. The taste can be almost wholly disguised by the addition of saccharin or

sassafras, or both. It can be put up in the following form:

Lime-water	12 ounces
Saccharin	½ grain
Sassafras	10 minims
Cudbear	15 drops

Sig: Use full strength as mouth wash several times daily.

The cudbear is simply a coloring matter, giving the solution a reddish color which tends to fade somewhat if allowed to stand indefinitely.

One of the most valuable mouth washes for general use in cases of periodontoclasia is a normal salt solution. Prinz refers to it as the safest and most efficient mouth wash that can be used. For clinical purposes, one-quarter of a teaspoonful of common salt to half a glass of warm water will be near enough to either a normal or physiological salt solution to meet the requirements of a mouth wash. It can be used several times daily to advantage in all such cases. In cases in which the salivary flow is subnormal, and stagnation follows, lemon juice, one ounce to eight ounces of water, is helpful. Its use will not only increase the quantity and alkalinity of the saliva, but the amount of the ptyalin also; because of its power to dissolve starch, this is an aid in cleansing the mouth and teeth.

WAXED DENTAL SILK

No matter how successfully the brush may have been used in an effort to clean the teeth, the interproximal surfaces will not have been brushed. And in those cases in which there is gingival recession, as is so often the case in periodontoclasia, triangular-shaped spaces are created, with their base at the gum line, making these surfaces of the teeth the most important to be cleaned, and the most difficult. The use of waxed dental silk in the cleansing of the teeth

is, therefore, as indispensable in these cases as is the brush. Its use should be made a part of the daily care of the mouth, for in no other way can the contact points between teeth be reached and cleaned. Not less than once a day is essential in all cases, and where there are abnormalities in contact relationships, it is desirable to use it after each meal. It is available in several widths, thus making it easy to choose one adapted to the needs of any case. Patients should be taught its use with the same care as that of the brush. It should perform the double function of removing food particles from between the teeth, and of polishing the proximal surfaces. This latter is accomplished by drawing the silk back and forth several times in each proximal space, bringing it to bear on both the mesial and distal surfaces. In those cases in which there is considerable gingival recession, an appropriate size of waxed silk tape should be used. The fear that one may injure the gingivæ in its use is unwarranted, providing the patient has been properly taught and follows instructions. Its use should always precede that of the toothbrush, as food particles thus loosened from between the teeth are removed by rinsing and subsequent brushing.

TOOTHPICKS

In those cases in which there is gum recession and wide-open spaces at the gum line, toothpicks in addition to the waxed silk can be used to good advantage. Not only are they useful as supplementing the dental silk in the removal of food particles, they are of even greater value in cleansing and polishing the tooth surfaces of these spaces. It is on these surfaces, just below and in contact with the gingivæ, that the soft, pasty material of the mouth is most apt to collect and most difficult to remove. The brush will not completely remove it, and the waxed silk as generally used

is not entirely successful. Dependence must be placed upon something that can be passed into these spaces and act as a scraping or polishing instrument. A toothpick of suitable size will not only do this and do it well, but at the same time be useful in stimulating the septal gingivæ. It should be passed back and forth, polishing each tooth surface in the space from the gum line to the contact point. Flat wooden picks can be used in cases where the spaces are wide enough to permit their use, and the quill toothpick in those of less width. Their use should be limited to those cases in which there is a gingival recession and triangular-shaped spaces. They have no place in the normal mouth, where their continued use may cause injury to the septal gingivæ.

(C) PROPHYLACTIC TREATMENTS

Prophylactic treatment, as its name implies, is a measure directed toward the prevention of disease, and as applied to the postoperative care of all cases of periodontal diseases, it represents the effort that is made by the dentist to coöperate with the patient in maintaining health in the tissues of the periodontium.[8] The frequency with which such treatments should be given will depend upon several factors, such as the natural cleanliness of the mouth; the amount of gingival recession; the efficiency or inefficiency of the patient's care, etc. These should all be taken into consideration, in deciding the frequency with which the patient should be seen for prophylactic treatments. All efforts should be directed toward increasing the patient's efficiency in the daily care of the mouth, and in decreasing the frequency of prophylactic treatments by the dentist. The daily care of the mouth, such as is well within the ability of any patient to give, is far more important in maintaining the periodontal tissues in health, than in fre-

quent prophylactic treatments by the dentist, without such coöperation on the part of the patient. Moreover, patients should be taught to depend upon themselves and not upon the dentist, for in no other way can the responsibility for the proper care of the mouth be placed where it belongs. It should be made clear to the patient at the beginning of treatment that he, and not the dentist, must be held responsible for the postoperative care of his mouth, since it is possible for the patient, through indifferent care, to nullify in large measure the most conscientious efforts of the dentist.

As a rule, it is impossible to determine at the conclusion of treatment just how often it will be necessary to see the patient for prophylactic treatments, as so much depends upon the patient himself. At the conclusion of the treatment, therefore, an appointment should be made three or four months later for the purpose of checking up on the operative treatment, and at the same time on the efficiency of the patient's home care. If satisfactory progress is being made, the next appointment can be at a longer interval. From two to four treatments annually should suffice for any case. These should include a thorough cleansing of the teeth such as has already been described; the reinstruction of patients in the part which they must play in the daily care of their mouths, and the reinspiring of them to persevere in well-doing. This latter is important, since it is the tendency of most patients to relax in their care as the need for it becomes less obvious, which it will as the case progresses toward health.

SUMMARY

It will thus be seen that the dentist's part in the postoperative care of his patients is an important one, consisting of the most painstaking instructions in the art of oral

hygiene; the prescription of the instruments by which this is to be accomplished with suggestions for their use; prophylactic treatments at regular intervals, and a never ceasing effort to inspire the patient to persevere in his personal care. This is the dentist's part in the postoperative program and one in which he must take the lead. It requires a profound conviction of its importance, and an enthusiasm to carry on in the face of discouragement. The patient's part, as outlined in the following pages, is no less important.

REFERENCES

[1] Fones, A. C., 1911, "The Gospel of the Toothbrush," *Dental Cosmos,* vol. 53, p. 443.

[2] Fones, A. C., 1927, "Mouth Hygiene," p. 286, Lea & Febiger, Philadelphia, Pa.

[3] Kennedy, C. M., 1921, "The Des Moines Toothbrush Lecture," *Journal American Dental Association,* vol. 8, p. 539.

[4] Lowry, A., 1920, "A Medicated Tooth Paste with Full Statement of Its Composition," *Journal Dental Research,* vol. 2, p. 565.

[5] McKenzie, C. M., 1917, "The Problem of Mouth Washes (Abstract)," *Journal Allied Dental Society,* vol. 12, p. 136.

[6] Prinz, H., 1916, "The Therapeutic Efficiency of Oral Preparations," *Journal Allied Dental Society,* vol. 11, p. 210.

[7] Spalding, Grace R., 1926, "Can Human Life be Prolonged by Dental Prophylaxis?" *Journal American Dental Association,* vol. 13, p. 1482.

[8] Stillman, P. R., 1917, "Prophylaxis for Pyorrhea," *Journal American Dental Association,* vol. 4, p. 156.

[9] William, M., 1917, "Saturated Solution of Salt as a Dentifrice," *Journal American Dental Association,* vol. 4, p. 1006.

[10] Hartzell, T. B., 1927, "Important Factors in the Etiology and Control of Periodontoclasia," *Journal American Dental Association,* vol. 14, p. 900.

CHAPTER XVI

THE PATIENT'S RESPONSIBILITY IN THE CARE OF THE MOUTH

Having been properly instructed in the home care of the mouth, it becomes the patient's responsibility to follow such instructions. Like the proverbial horse which anyone can lead to water but no one can force to drink, the patient can be properly instructed in the principles of oral hygiene without ever putting them into successful practice. In all such cases, the dentist should review his instructions for the purpose of locating responsibility for failure, and to make sure that he himself is not in some way at fault. In every case, he should encourage the patient to persevere, pointing out the ways in which he has failed, at the same time indicating the remedy. Simply to call attention to the patient's failures, without suggesting the necessary steps to be taken to correct them, is to discourage, rather than to help. One should always conclude his instructions with constructive and easily understood suggestions for improvement.

Under no circumstances should such patients be dismissed from one's practice, for there are none in greater need of care. The Golden Rule applies here as in every other aspect of life, and a kindly attitude toward the patient always brings the best results. The dentist should strive to overcome the handicap under which he may labor by redoubling his own efforts, and continuing his instruc-

tions and encouragement to the patient. In this way a measure of success is always possible, and while it may not be entirely satisfactory, it is always worth while just as half a loaf is always better than no loaf at all.

THE OBJECT OF PERSONAL CARE

The daily care of the mouth by the patient has for its object (*a*), the regular and efficient cleansing of the mouth for the purpose of reducing its bacterial content to a minimum; and (*b*), the stimulation of those biologic forces inherent in the tissues themselves, to the end that their resistance to disease be reinforced. These are of such importance in the daily care of the mouth, that to neglect either is to reduce by so much one's measure of success.[3]

This twofold object is to be accomplished largely by artificial means. This is unfortunate and would not be necessary if normal function provided the organs of mastication with the cleansing and stimulating effect it was designed to do, any more than it would be necessary for the laborer to resort to artificial means for the exercise of his body. Under the artificial and more or less unnatural conditions of modern civilization, the toothbrush has become a necessary and important agent in the prevention and treatment of all periodontal diseases. Its first purpose, assisted by the use of waxed silk which, as already indicated, should precede the use of the toothbrush, is to cleanse the mouth and teeth. In so doing, it reduces to a minimum the bacterial flora of the mouth, decreases the possibilities of infection, at the same time that it minimizes the mechanical and chemical irritation caused by the accumulation of tartar and food débris. In the second place the brush should, and will, if properly used, increase resistance and build up tissue tone through its stimulating influence upon circulation and cell activity. This, it should be remembered, is quite as

important as is its function of cleansing the teeth, and must always be given an important place in the patient's daily care of the mouth.

THE PRINCIPLES OF TOOTHBRUSHING

In order to accomplish this twofold function of mouth cleansing and gum stimulation, it is necessary that the patient be taught correct toothbrush habits, for almost no patient who has not been carefully instructed brushes his teeth properly.[6] The essential of any toothbrush technique is that it be simple, efficient and free from any injurious effect upon either the teeth or gums. These are the principles upon which all methods of toothbrushing must rest. Any method that combines all three, whether it be that of Fones,[2] Charters,[1] or Stillman,[7] can be recommended. It is foolish to assert that there is only one way in which to brush teeth, and that all teeth should be brushed in that manner. The only questions one should ask himself in considering any of the different methods advocated are, Is it so simple that any patient can understand and apply it in the daily care of his mouth? Is it effective in keeping the mouth clean and the gums stimulated, and does it do these essential things without injuring either teeth or gums? Any method that subscribes to all these conditions can be safely recommended. It is essential in all cases that these principles, as embodied in a definite technique, be taught the patient, for if left to his own devices, very little will be accomplished. This should be done largely by demonstration, as already pointed out, for in no other way will the patient get a clear understanding of what is meant by "proper methods of toothbrushing." To describe them by word of mouth, even when done with great detail, is not enough; they must be made the subject of demonstration.

A SIMPLE TOOTHBRUSH TECHNIQUE

It is also difficult to convey by the written word a description of any toothbrush technique that will be wholly clear to the reader, no matter how familiar he may be with the subject in general. They all seem complicated when described. In attempting, therefore, to outline one which the author has employed for years, he is not urging its adoption, or implying that there may not be other and better ways. Its purpose is to suggest a method by which the patient can care for his own mouth in a way that will be at once simple, effective and free from injurious effects. It also has the advantage of being easily understood, which is highly important. It is a mistake to make a toothbrush technique so complicated that not even the proverbial "Philadelphia lawyer" can understand and practice it. Simplicity of application is the first requisite to success in any method of toothbrushing.

Like everything else, the operation of brushing the teeth should be done according to some system; that is, it should proceed from start to finish in a definite way, though the method of procedure may vary to meet individual needs; it is principles rather than details that should be insisted upon.

BRUSHING THE TEETH

Having in hand a small brush of good quality, the mouth should be opened just enough to allow occlusal contact between the cuspids or incisors. In this way the muscles of the cheeks are relaxed and do not interfere with the free movements of the toothbrush. The brush should then be placed on the buccal surfaces of the upper molars of one side at right angle to their long axes. Without moving it from this position, it should be given a vibratory motion, which may be in a circular, up-and-down, or anterio-

posterior direction, meanwhile *forcing* the bristles into the embrasures between the teeth with considerable pressure. This should be done in such a way as to clean these surfaces as far as that is possible with a toothbrush, at the same time that it stimulates the septal gingivæ. When this is completed, and without removing the brush from the mouth, it should be lowered sufficiently to include the lower teeth in the same brushing technique. It is then brought forward to include the bicuspids, cuspids and incisors of both jaws as far as the median line. In this way, the buccolabial surfaces of all the teeth on one side of the mouth are brushed jointly and without removing the brush from the mouth. The brush may then be washed if desired, more dentifrice applied, and the same process repeated on the opposite side. By opening the mouth, third molars are brushed toward their occlusal planes in a sweeping motion, to include in the stroke the distobuccal surfaces. These surfaces will not be brushed unless it is done in a special way. In following this procedure, the buccolabial surfaces of all the teeth are brushed jointly, without necessarily removing the brush from the mouth. Having completed this part of the operation, the brush having again been washed, preferably in cold water, the mouth is opened and the brush brought to bear on the lingual surfaces of the molar teeth on one side, this time at a slight angle, with the bristles pointing toward the gingivæ. The same vibratory motion with pressure (usually on these surfaces in an anterioposterior direction) is repeated, until all the teeth in each jaw have been brushed from one third molar to the other. Because of the necessary separation of the jaws to permit the application of the brush to the lingual surfaces of the teeth, they cannot be brushed jointly as may be done on the buccolabial surfaces; those in each jaw must be brushed separately

Attention is next given to the occlusal surfaces of molars and bicuspids, which should be brushed with the same vibratory motion, in any direction most convenient to the patient. It should be remembered in the use of this technique that *pressure* on the teeth and gingivæ is a matter of supreme importance. As applied to the gingivæ, it intermittently empties and fills the capillaries as the brush is vibrated, in this manner effecting in a most salutary way the stimulation of the circulation in these tissues at the same time that it hardens and makes them more resistant.

STIMULATION OF THE GUMS

Having completed the brushing of the teeth, this is followed by the independent stimulation of the gums themselves with the toothbrush. By the time this part of the operation is in order, the brush will have become somewhat softened in use, which in its direct application to the gums is desirable, as there is then no danger of injuring them. As it is brought to bear on the buccolabial surfaces of the gums, the jaws are separated only enough to permit of contact in the incisor region. The side of the brush is then placed against the gums in the molar region of the maxilla, with its bristles pointing upward. It is then given a half rotation so as to bring the ends of the bristles against the gums with considerable pressure in a downward sweeping motion, always brushing from the gums toward the occlusal surface. This motion is repeated several times until all the gums in the upper jaw have been stimulated in this way. The brush is then reversed, bristles pointing downward and rotated in such a way as to brush the gums in the lower jaw upward, continuing this with pressure until the buccolabial surfaces have all been brushed. The jaws are then separated and the brush applied to the gums on the lingual surfaces of all the teeth and given a sweeping

motion toward the occlusal line. Considerable pressure should be brought to bear and a slight vibratory motion given the brush as it engages the septal gingivæ and moves occlusally. This is what might be called the sweeping method of gum stimulation. There are those who believe that it encourages the gingivæ to come more nearly into their normal positions, which, if so, is most desirable. In addition to its influence upon the circulation, it produces some friction on the surface of the gums which makes it necessary to exercise caution in the beginning.

Another method of gum stimulation is to use much the same technique as that employed in brushing the teeth; that is, the vibratory motion. The brush as applied to the buccolingual gums of the maxilla is pointed upward and then partially rotated so that the bristles are slightly bent on themselves. Then, with considerable pressure, it is given the same vibratory motion used in brushing the teeth. The same technique is repeated in the stimulation of the mandibular gums, with this difference, that the bristles are first given a downward inclination. If the brush is applied to the gums at a complete right angle as is done in brushing the teeth, and then vibrated under pressure, the ends of the bristles, if at all stiff, may prick the gums and cause irritation. Therefore they should be slightly bent on themselves as indicated above. This latter method of gum stimulation is preferred by the author not only because of its simplicity and efficiency, but because it is in line with the toothbrush technique. To teach another stroke motion in brushing the gums tends to confusion—something to be avoided as far as possible. The purpose of gum stimulation, as contrasted with gingival stimulation, is to arouse the circulation in the deeper periodontal tissues. In the correct brushing of the teeth, the gingival circulation is quick-

ened and the gingivæ themselves fortified and made more resistant to disease. In the prevention of periodontal diseases this may be all that is necessary. But in the post-operative care of cases that have been under treatment for periodontoclasia, the stimulation provided by an independent brushing of the gums, as distinguished from the gingivæ, is necessary since it animates the circulation in a way that is not done in gingival stimulation. This can be done by either of the methods above outlined. Whichever is used, it should be applied in such a way as to bring about intermittent pressure on the gums and gingivæ, and continued until a sense of glow pervades these tissues, which indicates a more active circulation. This is not brought about by the use on the toothbrush of some miraculously compounded "cure-all," as the promoters of these proprietary preparations would lead one to believe, but to the massage effect of the brush as it intermittently empties and fills the capillaries through the *pressure* which it exerts on the periodontal tissues when in action.

TIME OCCUPIED IN BRUSHING

This whole operation, including the brushing of the teeth, need not occupy more than three to five minutes once the technique has been mastered. There can, of course, be no set time. Irregular teeth and mouths in which there are fixed bridges will naturally require more time than will the uncomplicated case. The only rule should be an operation well done. This should be done at least twice a day, before breakfast and immediately after the evening meal. When the teeth are also brushed after breakfast and lunch, as is desirable, these two operations need not occupy more than two or three minutes each. To ask patients to spend ten or fifteen minutes twice a day in massaging the gums is,

generally speaking, unwise and unnecessary. One needs to be reasonable in this as in everything else.

Other methods for stimulating the gums other than by the use of the brush may be employed if preferred, such as the finger, rubber brushes, etc., though it should be admitted that, generally speaking, they are less efficient. In an effort to make the procedure as simple as possible, it is generally unwise to complicate it by the use of too many different implements. Since a brush is indispensable in toothbrushing, it is desirable that the stimulation of the periodontal tissues be done by it also.

SUMMARY

This, in brief, is what is meant by the patient's part in the regular and postoperative care of the mouth. It includes the use of dental floss at least once a day; the regular and efficient brushing of the teeth not less than twice daily, before breakfast and after dinner, preferably four times a day; the stimulation of the gums by a definite technique each time the teeth are brushed; and in certain cases, the use of toothpicks. It also includes the carrying out of any dietetic instructions which may have been given. When these rules are intelligently followed, the results will be so gratifying as to make the treatment and postoperative care of periodontal diseases one of the most satisfactory features of dental practice.

REFERENCES

[1] Charters, W. J., 1919, "The Ideal Method of Brushing the Teeth and Standardizing the Toothbrush," *Journal American Dental Association*, vol. 7, p. 155.

[2] Fones, A. C., 1915, "Instructions for the Home Care of the Teeth," *Dental Items of Interest*, vol. 37, p. 365.

[3] Rauh, S. J., 1919, "Toothbrush Exercise," *Pacific Dental Gazette*, vol. 27, p. 365.

[4] Sarazan, J. J., 1920, "The Toothbrush and Methods of Brushing the Teeth," *Journal American Dental Association*, vol. 7, p. 155.

[5] Smith, T. Sidney, 1921, "Some Essential Facts in the Use of the Toothbrush," *Dental Items of Interest,* vol. 43, p. 72.

[6] Spalding, Grace R., 1914, "Instructions for Oral Prophylaxis," *Dental Cosmos,* vol. 56, p. 318.

[7] Stillman, Paul R., 1924, "Physical Culture for the Gums," *Dental Cosmos,* vol. 56, p. 1042.

[8] Tishler, B., 1920, "Prophylaxis," *Journal Dental Research,* vol. 2, p. xxlx.

CHAPTER XVII

THE PREVENTION OF PERIODONTAL DISEASE

The prevention of disease in the tissues of the periodontium is a matter of such importance as to justify its separate consideration, even though, in doing so, one runs the risk of repetition. Because of the tendency which these diseases possess of breaking down and destroying the periodontal tissues, no treatment, no matter how skillful, can ever be wholly satisfactory. At best it can only be a partial success, for in a very real sense, disease in these tissues creates a condition that is in a measure irreparable. Because this is true, it would be difficult, if not impossible, to overestimate the value of prevention in the field of periodontology. All are agreed that the prevention of disease is vastly more important than is its cure, but since no one can accurately measure the effects of disease, it logically follows that the value of prevention can likewise never be wholly known. This is unfortunate, for it places a premium upon cure, instead of placing it upon prevention, where it belongs. This explains also why it is that the surgeon occupies a more exalted position in public estimation, and is more highly rewarded, than is the prophylactician. There is something in the successful treatment of disease that appeals to the imagination. It has in it an element of the dramatic. The whole world applauds when the surgeon rescues his patient from the bondage of disease. It is something positive, something tangible; its worth can be measured. And yet, in many instances, no matter how skillfully the surgeon may do his work, he can never wholly

restore to the patient that which has been lost through disease. Its progress, as in certain diseases of the periodontium, is frequently attended by conditions that are irreparable, and which will continue as lifelong handicaps, no matter how successful has been its treatment. These are the scars, so to speak, that continue to remind one of the vastly greater value of prevention. For these reasons, if for no others, it is distinctly worth while to consider every known resource in the prevention of periodontal diseases with a view to their application in daily practice.

DIFFICULTIES IN PREVENTION

At the outset it must be admitted that complete success in the field of prevention is not possible—a fact that may be discouraging, but which should not discourage. As already stated, there are factors in the etiology of certain periodontal diseases, notably periodontoclasia, that are at present not wholly understood, and which cannot therefore be brought under complete control. Especially is this true of that type of periodontoclasia having a systemic background and by some believed to be associated with abnormalities in diet and nutrition. Fortunately, such cases are not in the majority. And even in these, local preventive measures are always helpful and in some instances may actually be effective.

FIG. 52.—Hypertrophic gingivitis due to lack of care. Cleanliness and gum stimulation would have prevented this condition. Male patient.

It is, however, in the realm of local conditions which include such factors as oral sepsis, disfunction, lowered resistance, bacterial accumulations, etc., that the largest measure of success must be looked for in the prevention of periodontal diseases.[6] These can, to a considerable

extent, be brought under control, and in doing so a very large percentage of all periodontal diseases can be prevented or checked in their incipiency. (Fig. 52.) All efforts in this field are directed toward the accomplishment of two things: stimulating circulation in the periodontal tissues in order that their resistance to disease may be increased; and in reducing to a minimum, through prophylactic treatment and toothbrushing, the bacterial flora of the mouth.[3]

THE STIMULATION OF PERIODONTAL CIRCULATION

This is to be brought about by exercise—natural and artificial. The normal exercise of any tissue augments its blood supply, increases its vitality, and fortifies it against disease. If the organs of mastication could be given the stimulation of vigorous exercise in the function of mastication, many of the ills that now attend them would cease to exist. Malformation of the jaws, irregularities of the teeth, impacted and missing teeth, can all be directly traced to the degenerating influence of relative disuse. This factor of disfunction plays a large part in the diseases affecting these tissues, though in this aspect of the problem, diet and nutrition have also to be considered. Of first importance, therefore, in the prevention of periodontal diseases, are well-nourished tissues, and the natural way to accomplish this is through function.

FUNCTIONAL STIMULATION

Patients, especially young patients, should be encouraged to use their teeth and jaws more vigorously than they do in the mastication of their food. While the modern preparation of foods offers little incentive to vigorous chewing, they could and should be masticated far more thoroughly than is done by most people. This would be of

great advantage, not only to the teeth and jaws, but to the general health as well. People have formed the habit of bolting their food, which is largely responsible for that so-called American disease of dyspepsia.

Therefore, the dentist in his care of his patients, and especially in his efforts at prevention, should instruct them in the importance of thorough mastication and encourage them to include in their dietary such foods as require vigorous chewing. Those of them who are parents should be advised to pay especial attention to the masticatory habits of their children, in order that they may early form the habit of correct mastication, for this can be made as much a habit as is food bolting.

ARTIFICIAL STIMULATION

Functional stimulation of the periodontal tissues through the act of mastication should be supplemented by artificial means. This would not be necessary if the need of thorough mastication were enforced by the daily use of raw, coarse food. But since this is not altogether possible, it becomes necessary to resort to artificial stimulation for the purpose of increasing periodontal circulation. As already pointed out, the most satisfactory way in which to accomplish this is by the proper use of a toothbrush. And since the technique for its use in this connection has already been described in the chapter on postoperative treatment, it will not be necessary to repeat it here. It should be said, however, that advice as to the best methods of artificial stimulation should be made a prominent part of all instructions to patients, inasmuch as it and functional mastication are the only means by which periodontal circulation can be stimulated. Well-nourished tissues are the most effective barrier that can be raised against disease.

THE REDUCTION OF BACTERIA THROUGH PROPHYLACTIC TREATMENTS

As already stated in the chapter on etiology, bacteria play a conspicuous rôle in all periodontal diseases. In Vincent's infection and periodontoclasia, they represent the controlling factors, for neither of these diseases as they are clinically known could exist without the presence of bacteria in the mouth. This being true, their control is a matter of supreme importance in any effort at prevention. The organisms causing these diseases gain entrance to the periodontal tissues through the gingivæ and the cemento-enamel attachment at the floor of the gingival crevice. The gingivæ are, so to speak, the gateway to all the diseases of the periodontium. For this reason, it is essential that they be maintained in health, for by so doing the door to disease is effectively closed. (Fig. 53.)

FIG. 53.—Chronic gingivitis of a hemorrhagic type. Many of the gingivæ are almost purple in color. Due to lack of mouth hygiene and gum stimulation.

THE NORMAL GINGIVÆ

In order to obtain a clear conception of their function and an accurate picture of their appearance in health, one should study the gingivæ of children and young adults. The dentist has become so accustomed to dealing with these tissues in disease as scarcely to know what a normal gingiva looks like. And one of the reasons for this is, that among adults there are few patients in which they are entirely normal.

In health they are as a rule elevated above the alveolar crest to the extent of a millimeter or more. As pointed out

by Gottlieb, this relationship is constantly changing and varies with different individuals. Between the gingivæ and the cervical enamel is the gingival crevice (sometimes called gingival sulcus, gingival space, gingival pocket, etc.), bounded at the cemento-enamel junction by the attachment of the gingival group of pericemental fibers. In health, the gingivæ terminate in a thin knife-like edge which hug the cervical enamel in such a way as to protect the pericemental attachment against injury.

THE GINGIVÆ IN DISEASE

If, as a result of irritation, these projecting tissues, so admirably suited to their purpose, become inflamed, their protective function is more or less seriously impaired. They no longer permit food to glide over their external surfaces in mastication without injury, but certain portions of it are forced into the gingival crevice adding to the already existing irritation. As a rule, this does not occur symmetrically around individual teeth or throughout the mouth. It will be further observed that irritation occurs in those regions of the mouth that are least accessible and therefore least clean, emphasizing in this way the importance of oral hygiene in maintaining these tissues in health.

ORAL HYGIENE

Much has been said and written in the last twenty years about the influence of oral hygiene as a preventive of dental caries. "A clean tooth never decays" has become almost a proverb. And there can be no doubt that there is much truth in that statement. According to Bunting,[1] dental caries "is a specifically infective disease." This being true, the cleaner the teeth the greater their freedom from dental caries. "Cleaning teeth," as used in this con-

nection, has always meant the polishing of the exposed tooth surfaces in the belief, justified from experience, that such polished surfaces are less liable to decay.

THE DENTAL HYGIENIST

It was out of this conception of the oral hygiene movement that the dental hygienist had her origin. If dental caries was to be prevented, teeth must be kept clean and their surfaces polished. The problem also had its economic aspect. It was an important health service and one that should be placed within the reach of all, hence the creation of a new profession, the members of which are known as dental hygienists.

Their duties, as regulated by law, are to clean and polish the exposed surfaces of the teeth for the purpose, chiefly, of inhibiting tooth decay. If "by their works ye shall know them," they have long since justified the profession of dental hygiene. They have done their work well and played an important part in the prevention of tooth decay. Too much credit cannot be given them for the efficiency with which they have done their work, and for the high standard of professional ethics which they have always maintained. If the prevention of disease is the highest service that can be rendered, then the dental hygienist is entitled to take high rank, for her every professional act is directed to that end.

ORAL HYGIENE AS APPLIED TO PERIODONTIA

But oral hygiene as applied to periodontia is quite a different matter. As practiced by the dental hygienist and by many dentists, it undoubtedly is an aid in the inhibition of dental caries, and of importance, as far as it goes, in the prevention of periodontal diseases. Unfortunately it does not go far enough to meet the requirements of the latter.

It is not what is on the tooth, but what is under the marginal gingivæ and in contact with them that is the chief local source of disease in the periodontal tissues. The exposed surfaces of the teeth may be as clean and polished as the trained hand of the dental hygienist can make them, at the same time that the gingival crevice is filled with a stagnating mass of decaying food particles, bacterial accumulations, serumal calculus, etc. The unfortunate and disconcerting feature of this situation is, that these conditions can and do exist, with very little clinical evidence of that fact. In incipient cases where the lesions in the gingivæ are in their early stages, there may be no clinical evidence whatever to indicate this, at least none to be seen by the casual observer. For these reasons, the dental hygienist can hardly be blamed if in her care of adult patients she fails to note these early symptoms of periodontal disease. Unfortunately, she is sometimes placed in the position where she is expected to be both prophylactician and diagnostician. It not infrequently happens that in offices where she is employed, it is her duty to send for patients at regular intervals, at which time she cleans their teeth, examines them for caries and, finding none, dismisses the patient without his having seen the dentist at all. She is not qualified by training or experience to pass upon the health or disease of the periodontal tissue, yet in the position in which she frequently finds herself, she is in effect actually doing this. This is not her fault, but the fault of the system which has grown up through her employment. Because of her incapacity to measure up to these obligations, no adult patient should be entrusted to her care until after the family dentist has first made an examination to determine whether it is a case requiring only the cleaning and polishing of the exposed surfaces of the teeth. In that event, the dental hygienist can do all that is necessary.

There are few adult patients, however, of which this is true. In childhood, when the gingivæ are performing their normal function of protecting the cemento-enamel attachment at the floor of the gingival crevice, and before disease has robbed them of their protective function, the polishing of the teeth is all that may be necessary. But in adult life, when almost no patient possesses a perfectly healthy periodontium, where there is scarcely a gingival crevice in the mouth in which there is not some break in the cemento-enamel attachment, cleaning the teeth in the way in which it is usually done is wholly inadequate. (Fig. 54.) For these reasons it will be obvious that oral hygiene, as applied to periodontia, has a connotation quite different from that usually given it. It includes all that is usually meant by that term, and begins where it leaves off. It stands for preventive dentistry, and is aimed at the removal from the mouth of all those factors which make for disease, such as salivary and serumal tartar; food particles from around and between the teeth; mucin plaques; roughened and unpolished surfaces; overhanging edges of fillings, bacteria, filth, etc., in order that the teeth and their supporting tissue be maintained in health as far as that is possible by the application of local measures.

Fig. 54.—A moderately advanced case of gingivitis with early pocket formation in a young patient. Note bad hygiene.

PROPHYLACTIC TREATMENTS: THEIR OBJECT

How this is done is usually summed up in the term "prophylactic treatment," an operation of such importance in the care of the mouth as to justify a detailed description. And while its application is here made to apply to the prevention of periodontal diseases, it is quite as applicable to their postoperative treatment.

After a preliminary examination to ascertain what the general condition of the mouth is, and before any polishing of the teeth is done, the gingival crevice of each individual tooth is explored throughout its circumference with some of the curettes or planes designed for that purpose. This has for its object the removal of the contents of the gingival crevice, usually consisting of a pasty mass of food débris, and, in some instances, a more or less hard serumal tartar. The amount of such material that can be crowded into a single gingival crevice, with little or no visible evidence of its presence, is often a matter of the greatest surprise. Mouths that appear on occular examination to be clean and healthy, will often be found to be anything but that when explored in this way. For this reason nothing can be taken for granted. No examination of the mouth, no cleaning of the teeth, can be regarded as complete that does not include the exploration of the gingival crevice and its treatment, if necessary.

FIG. 55.—Very early manifestations of periodontal disease. Note gingivæ about upper right lateral, lower left cuspid and first bicuspid, and lower right first cuspid. These symptoms are so slight as to be overlooked in most cases. A few minutes spent in treatment at this stage of disease will save a beautiful set of teeth. Every hopeless case of periodontoclasia was at one time in this stage of development, and could have been cured in a few minutes of treatment.

EARLY POCKET FORMATION

The gingival crevice should also be examined to ascertain whether pocket formation is beginning. And here another surprise awaits the inexperienced. In many cases where it is least expected, it will be found that solution of continuity has already taken place in the floor of the gingival crevice about certain teeth. (Fig. 55.) This is especially

true of adults, even young adults. For these reasons, as already indicated, it is unwise to entrust the care of adult patients to the dental hygienist, and as a rule it should never be done. If there be indications of disease, its treatment is naturally outside her province. While this may limit her field of activity, it in no way reflects upon the dignity of her work or the usefulness of her calling, any more than it reflects upon the dentist not to be able to do a tonsillectomy. The dental hygienist is engaged in the far more important work of prevention, and if in her work among children, where she is so well fitted to serve, she be permitted to do her duty, there will be fewer adults requiring the services of the dentist. She will be stopping at the source that long train of evils comprehended in the term, periodontal diseases.

CLEANING TEETH

Having completed this part of the prophylactic treatment, the next step includes the cleaning of the teeth. This is also important and has for its purpose the removal of stains, the polishing of the teeth, and the reduction of mouth bacteria. In cases of gingival disturbances of any kind, it should include their treatment.

A preparation of silex, sold under the trade name of "Politor" and prepared by the W. M. Sharp Manufacturing Company, Inc., Binghamton, New York, is a safe and efficient polishing agent. Pumice stone should not as a rule be used, as it is too harsh in action and does not leave a highly polished surface. The silex preparation is placed in a proper receptacle and made into a stiff, creamy paste by the addition to it of some pleasant mouth wash. Liquor antisepticus U. S. P., because of its alcoholic content and cool and refreshing taste, will be found to be satisfactory. A solution of this kind is used instead of water for the

following reasons: First, it reduces to a minimum the tendency which all polishing preparations of this nature have of becoming "stringy" and so spattering when used; and second, by flavoring it with an agreeable tasting mouth wash, the operation of polishing the teeth is made less unpleasant. A disclosing solution is also necessary. There are many different formulæ available, and any one to which the operator is accustomed may be used. In all cases in which there is gingival inflammation, some treatment is desirable. A preparation that will serve the double purpose of disclosing stain and treatment for the gums is tincture of iodine and aconite root, equal parts. The proper amount is placed in a small Dappen glass and diluted slightly just before using. Drawing back the lips, the teeth and gums of one jaw are painted with this solution, and while it still bathes them, an inverted cone-shaped brush, charged with the polishing agent and driven by the dental engine, is brought to bear upon all accessible surfaces of the teeth, always brushing from the gums toward the occlusal surfaces. With this, all the buccolabial surfaces are brushed and such of the lingual surfaces as can be reached. It is then replaced by a wheel-shaped brush, which is brought to bear on all occlusal and lingual surfaces, and upon such other surfaces as may not have been previously brushed by the cone-shaped brush. The same operation is repeated on the other jaw, after which the mouth is carefully sprayed. By the use of these brushes (rubber cups and disks can be used, if preferred), the grosser stains and bacterial plaques are removed in a fraction of the time that would be required to do it by hand, and quite as efficiently. This may sound like heterodoxy, for there are those who feel that this should all be done by porte-polishers. The purpose of the operation is the cleansing and polishing of the exposed surfaces of the teeth, some part of which can be

better and much more expeditiously done with engine-driven instruments such as brushes, rubber cups, etc., than is possible with hand-polishing instruments. When properly done, there need be no injury to either teeth or gums. Following the use of the brushes, the teeth and gingivæ of each jaw are again painted with the iodine solution, and carefully gone over with porte-polishers. It will usually be found that some plaques have eluded the use of the brushes; these are made visible by the disclosing stain and can be removed by the hand polishers. All the teeth should be gone over in this way. Waxed silk tape, the size of which is regulated by the case in hand, is charged with the polishing agent and drawn back and forth between each tooth for the purpose of polishing these surfaces. Strips may occasionally be used, but should, as a rule, be limited to the proximal surfaces. If too free use be made of them in polishing the cervical margins, these may become sensitive. It is important, however, that all roughened surfaces, all overhanging edges of fillings, in fact, anything that interferes with the efficient cleaning of the teeth, be removed. Such conditions act as holding media for bacteria and should not be tolerated.

CARE OF DENTAL BRUSHES

After use, these dental brushes should be removed by the dental assistant and thoroughly washed, using a hand brush to aid in removing the remnants of the polishing preparation, after which they are submerged in a solution of alcohol and allowed to remain for several hours. They are then removed and made ready for use later. The alcohol evaporates quickly and leaves them dry and hard. As soon as they show wear, they should be thrown away. They cannot be boiled without injury and discoloration of the bristles.

MOUTH EXAMINATION

There is nothing in the prevention of all dental diseases more important than a thorough and comprehensive examination.[9] Unfortunately, there is no one point in the entire practice of dentistry at which more failures occur than in the examination of the mouth and teeth as it is usually done. Indeed, it is often regarded as of such little importance that the dental hygienist is permitted to make it. As frequently made by the dentist, it consists of little more than a perfunctory examination for caries. There are two explanations for this: The first is that the examination is not comprehensive enough. It is usually limited to an examination of the teeth for caries; to the need of prosthetic restorations of one kind or another, possibly to deposits of salivary tartar, with almost no attention being paid to diet or conditions of the general health as they may affect the local situation. The important matter of focal infections as they are represented by periodontal diseases and pulpless teeth are frequently not included in the examination. In the second place, the examination is not thorough enough. Even in its limited sphere it is incomplete. Caries are not always discovered in their incipiency, as they should and can be. Periodontoclasia is frequently well advanced before it is discovered and the general hygiene of the mouth is too often overlooked altogether. In a word, the examination of the mouth and teeth as it is usually made is very far from being satisfactory to anyone, when, in fact, it should be the most important in the dental care of the patient. To undertake such care without a complete and thorough examination on which to base diagnosis and treatment, is like making deductions from a false premise: the results are bound to be wrong. There is no way in which the confidence of the patient can be so easily

lost as to tell him that there is nothing wrong with his teeth, and to have him discover later that several carious places have been overlooked, or that his teeth are becoming loose from periodontoclasia. When this happens, the family dentist has usually lost a patient with no one to blame but himself. One of the most embarrassing questions asked of the periodontist, by the patient who presents for the first time, with a more or less advanced case of periodontoclasia is, "Why is it my dentist has not discovered this long since?"

There is no time when the examination of the mouth and teeth can be made to better advantage than at the conclusion of a prophylactic treatment. This should include a search for caries, open contact points, pulpless teeth, oral sepsis, malocclusion and the condition of the periodontal tissues. The matter of lost teeth and their possible restoration should also be given consideration, as should the question of diet, general health, etc. Indeed, it should include everything which has for its purpose the prevention and treatment of disease within the oral cavity. In all this a full radiographic examination is of first importance, since no examination of the mouth and no dental care of the patient can ever be regarded as complete that does not include a radiographic examination of the jaws and teeth at regular intervals.[4, 6] In no other way can one know what is going on within the tissues of the periodontium, upon the health of which depends the usefulness and health of the teeth themselves. In the detection of proximal caries in their early beginnings, the radiogram is also exceedingly useful and should be made a part of the routine examination whenever possible. It is realized that this cannot always be done, that for economic or other reasons, there are patients who will not accept the advice given. The patient should, nevertheless, be informed that no examina-

tion can be regarded as complete that does not include a radiographic survey, and that if this is not done, the dentist cannot be held responsible for possible failures. It is not in such instances, however, that most failures are made, but in omitting to make radiographic examination in those cases where it could and should be done.

DIET IN PREVENTION

Having included in one's examination a careful scrutiny of local conditions, one should next turn his attention to those factors which lie outside the domain of purely local conditions. And this includes diet. If abnormalities in diet are capable of setting up retrograde changes in the alveolar tissues independent of local conditions, then it is important, in the prevention of periodontal diseases, that this fact be taken into consideration. Inquiries regarding the patient's dietary habits should be made and such instructions given as may be necessary to correct them. Since the diffuse atrophy associated with certain cases of periodontoclasia seems to be associated with calcium deficiency or disturbance in the acid-base balance, it is of the utmost importance, in the prevention and treatment of these cases, that such factors be eliminated by dietetic reforms. These would include the adoption of a diet rich in calcium such as is found in dairy products, leafy vegetables and fruits. For further information on this subject, the reader is referred to "The Newer Knowledge of Nutrition" by E. V. McCollum, "Rational Diet" by Otto Carque, and to the brief chapter on diet in the present volume.

THE GENERAL HEALTH

It was said that in the examination of the patient for the purpose of preventing diseases affecting the dental structures, one should take into consideration those factors

which lie outside the domain of purely local conditions, which, in addition to matters pertaining to diet, must include inquiries concerning the patient's general health. This should be done for two reasons: first, because conditions of the general health may unfavorably influence the prevention and treatment of dental diseases, and second, because, in so doing, the dentist may render a far more important service than would be possible were he to confine himself to purely dental considerations. Patients do not, as a rule, see their family physician with the same regularity that they do their dentist, which fact places upon the latter a greater responsibility to be alert to the patient's physical welfare. When such inquiry reveals suspicious symptoms, the patient should be advised to consult his physician, and when dental conditions warrant it, they should be reported to the physician for his information. It is only through coöperation in this way that the best interests of the patient can be served.

SUMMARY

The dentist in his efforts to prevent diseases of the teeth and their investing tissues, should, with the coöperation of the patient, do everything possible: first, to promote active circulation in these tissues, in order that there may be erected the most effective barrier possible against their invasion by bacteria; second, by regular prophylactic treatments and intelligent home care, to maintain at all times a high standard of mouth hygiene; third, to see to it that patients are advised in the important matter of diet; and fourth, by inquiry regarding the patient's general health, to be in a position to understand its relation to local conditions, and the possible need of enlisting the coöperation of the physician. When these very simple rules are observed, it is entirely within the truth to say (and this is borne out

by experience) that most of the dental diseases which are now so common as to include most civilized peoples, could be prevented altogether or checked in their incipiency. And there is no higher service to be rendered in the practice of dentistry.

REFERENCES

[1] Bunting, R. W., 1929, "Oral Pathology," p. 188, Lea & Febiger, Philadelphia, Pa.

[2] Bunting, R. W., 1923, "Rules for the Prevention of Periodontal Diseases," *Journal American Dental Association,* vol. 10, p. 145.

[3] Merritt, A. H., 1928, "Preventive Periodontia," *Journal Dental Research,* vol. 8, p. 141.

[4] Pollia, J. A., 1928, "Fundamental Principles of Alveolo-Dental Radiology," *Dental Items of Interest,* vol. 50, p. 784.

[5] Symposium, 1928, "The Principles of Preventive Periodontia," *Journal American Dental Association,* vol. 15, p. 1072.

[6] Warner, G. R., 1926, "Radiography and Diagnosis," *Journal American Dental Association,* vol. 13, p. 1710.

CHAPTER XVIII

SENSITIVE TEETH: THEIR DIFFERENTIAL DIAGNOSIS AND TREATMENT

It frequently happens that teeth from around which the gingivæ have receded become sensitive at times to friction from the toothbrush, to anything that is sweet, and to changes in temperature. Such conditions may also alternate with entire freedom from symptoms. These sensitive areas are located on exposed roots or cervical portions of the tooth where the dentine is exposed, and usually on the buccolabial surfaces. The exposure may be so slight as to be scarcely noticeable, or it may involve half the root surface. It may or may not be associated with periodontoclasia. Anything that brings about exposure at these points is the predisposing cause, since it is only such teeth that become sensitive. Not all teeth with exposed dentine or cementum become sensitive, nor in those cases in which it may occur does it affect all teeth alike. Some may be exquisitely sensitive, while others in the same mouth, with an equal amount of exposure, will remain permanently insensitive.

The manner in which sensation is transmitted through the dentine to the pulp is not clear; the presence of nerve filaments in the dentine and cementum is the probable explanation.

SENSITIVE AREAS

There are three areas in the structure of each tooth, not including the pulp, that are by nature exceedingly sensi-

tive: [1] These are (*a*) the dento-enamel junction; (*b*) the granular layer of Tomes; and (*c*) the interglobular spaces. The sensitiveness of the dento-enamel junction is due to the increased amount of noncalcified organic matter always present at this point, and caused by the branching of the dentinal tubules in the dentine in this area. Its sensitiveness to the use of an instrument is a matter of the most common observation among dentists. This is because of its very intimate relation to the dental pulp.

THE GRANULAR LAYER OF TOMES

This is limited entirely to the root surface of the dentine. It is a noncalcified and highly sensitive zone, located on the periphery of the root immediately beneath the cementum. The so-called "sensitive necks" of teeth owe their existence in large measure to the exposure of this granular layer. In health it is normally covered at the cervix of the tooth by a thin layer of cementum, which in turn is protected by the gingiva. In cases of recession, the cementum, which at this point is not thicker than a human hair, is frequently removed by the toothbrush, in this way exposing an exceedingly sensitive area of the tooth. Since, however, this granular layer is found only on the periphery of the dentine of the root, and rarely extends into the crown of the tooth, its sensitiveness becomes evident only in those cases in which exposure from gingival recession occurs. It is the exposure of this area which probably explains most of the cases that present themselves for treatment of sensitive teeth.

THE INTERGLOBULAR SPACES

The presence of interglobular spaces in the dentine is indicative of imperfect calcification. Such teeth, generally referred to as "soft teeth," are always more sensitive than

are the so-called hard or sclerotic teeth. When for any reason the dentine of such teeth becomes exposed, it is often extremely sensitive, due to the presence in it of non-calcified organic material. The less perfect the calcification of the tooth, the larger will be the area of interglobular spaces. It is this type of tooth that is always more or less sensitive to any form of operative interference.

These interglobular spaces are irregularly distributed in the dentine, being found in greatest abundance in a direction running parallel with the dento-enamel junction. For this reason their exposure is dependent in large measure upon the loss of enamel. Gingival recession without enamel loss would not be likely to expose this area, and therefore any sensitiveness which might be present would be due to some other cause. It is in those cases of abrasive gingival recession in which there is a considerable loss of tooth substance, including enamel, that these interglobular spaces become exposed. It is this latter type of recession that is most apt to be sensitive, for it exposes at one and the same time the dento-enamel junction, the interglobular spaces and that area which is the most sensitive of them all, the granular layer of Tomes.

EFFECTS OF ACIDS

When to the natural sensitiveness of these areas is added their exposure to the irritating influence of acids so often met with in the mouth, every element making for highly sensitive tooth surfaces will be present. Bacterial fermentation in many instances is accompanied by the liberation of lactic acid which greatly increases the irritability of the protoplasmic contents of the dentinal tubules. This explains why it is that the excavation of a tooth involved in active caries is always more or less painful and doubtless

explains also why it is that exposed root surfaces are sensitive at one time and not at another. The irritating effect of acids should therefore always be considered in dealing with these cases.

It was Nature's design that any part of a tooth not covered by enamel should be protected from the secretions of the mouth by the investing tissues. When these are destroyed, the exposed surface is thrust into an environment that is entirely foreign to it and for which it is wholly unfitted. When this occurs suddenly, as in the operation for gum resection already described, there is always more or less sensitiveness, especially to changes in temperature, which may continue with some modification indefinitely. This is one of the arguments against this operation. When the exposure is gradual, as is usual in most cases of gingival recession, this sensitiveness is less marked and, in most instances, absent altogether. This is probably due to the inherent sensitiveness or insensitiveness of the teeth and to the nature of the oral secretions. Fortunately, because of Nature's adaptability, teeth with exposed root surfaces as a rule tend to adjust themselves to their changed and unnatural environment. If this were not so, any operation which involved loss of gum tissue would in every case be contraindicated. Their adjustment, however, in cases of gum recession, if at all extensive, is usually only partial, which should make gum resection the last resort of the periodontist.

TYPES OF SENSITIVE TEETH

There are two more or less distinct types of sensitive teeth that have their origin in periodontal abnormalities, which, though their etiology and pathology are the same, are quite different in their symptomatology and treatment. For this reason, it is essential that a differential diagnosis

be made in order to determine which type one is dealing with.

The *first* type is characterized by surface irritation. The exposed area is acutely sensitive to touch, as when the toothbrush, finger nail, or steel instrument is brought to bear on it. It may also react to anything that is sweet, acid or saline. In addition, this type of case may be sensitive to thermal changes, though this latter symptom is usually not marked, and is of secondary importance in diagnosis. When present, it represents a merging of the two types, in the first of which surface sensitivity is the chief diagnostic symptom, while reaction to temperature changes characterizes the second. When the latter symptom is present in the first type, it is usually due primarily to irritability of the exposed area, rather than to pulp disturbance. Both factors may be present in a given case, though this is the exception and not the rule.

TREATMENT

Since it is impossible to get the gums or gingivæ to grow back and cover these exposed and sensitive areas which would be the logical treatment, it is necessary that they be desensitized in some way. This is usually best accomplished by cauterization through the application of drugs. Of these, formaldehyde, full strength, is perhaps the most satisfactory when properly used. To cauterize, place a few drops in a small Dappen glass, and, having protected the mouth with a napkin, take an orangewood point, dip it in the formaldehyde, and having absorbed all excess by touching it to a napkin, apply the wet point directly to the sensitive area and rub it vigorously. Repeat this several times, spending altogether three to five minutes in the operation, or until all sensitiveness has disappeared. Usually one such treatment is sufficient. If not, it should be

repeated. In very sensitive cases, there is usually a little pain on first application due largely to the friction produced by the orangewood point, but this disappears as the rubbing continues. It is this *rubbing* of the formaldehyde into the tooth itself that is an important part of the treatment. Its application on cotton and drying it with hot air, as is sometimes advocated, is inefficient and in most instances of no value whatever. Care should always be exercised in its use to prevent its coming in contact with the gums as it may cause sloughing, though there is not the slightest danger of this if used as directed. When properly applied, it is a most efficient though not infallible remedy. Zinc chloride used in the same way is often helpful in the treatment of these cases, though as a rule less efficient than formaldehyde. If for any reason the latter does not give results, zinc chloride is the next choice. Its use frequently yields excellent results.

THE USE OF SILVER NITRATE

In those cases in which the application of formaldehyde or zinc chloride is difficult, and where staining does not have to be considered, nitrate of silver may be employed. A very satisfactory way to apply this is to have the druggist reduce the crystals to a fine powder, and having protected the mouth by napkin or cotton rolls, dip the wet end of an explorer in the powder and carry it directly to the area to be treated. The wet surface of the tooth will liquefy the powder as soon as it is brought into contact with it. Several applications of the silver nitrate can be made at the one treatment and the whole precipitated by the application of eugenol before the removal of the napkin. The eugenol should be allowed to remain in contact with it several minutes. The advantage of this method of applying nitrate of silver is the ease with which a definite amount

can be applied directly to the point desired and nowhere else. In powder form, it will not deteriorate as it will if held in solution and it is more efficient. It is not, however, as efficient as formaldehyde and it has the added disadvantage of discoloring. Nevertheless it has its place in the treatment of these cases, and when limited to those areas where other preparations cannot be conveniently applied, as in approximal surfaces of teeth, it will be found to be useful. When applied to surfaces where incipient caries is present, and the application repeated several times and precipitated, it will arrest further progress. Because it leaves no stain, and in most cases is effective, formaldehyde is the treatment par excellence for all cases in which it can be conveniently applied.

DESENSITIZING PASTE

A preparation containing paraform has been highly recommended for the treatment of sensitive areas at the necks of the teeth. It is composed of the following ingredients which in preparing the paste should be carefully mixed:

Paraform	3 parts
Orthoform	2 parts
Thymol	0.25 part
Cotton and paraffin	1.75 parts
Soft paraffin	7 parts

In using it, cleanse the abraded surface and wash it off with warm alcohol. Dry it with warm air and apply a *very small* amount and smear it over the surface. This is then sealed by covering the entire surface with a thin mix of oxyphosphate of zinc cement. It should not be allowed to remain more than forty-eight hours. Paraform is a potentially dangerous drug when applied too near the pulp or when used in too large quantities.[2]

POSTOPERATIVE TREATMENT AND SENSITIVE TEETH

Inasmuch as acids due to bacterial activity are often a factor in causing hypersensitiveness of exposed root surfaces, their neutralization would seem to have a logical place in treatment. In the postoperative care of these cases, especially those in which the exposure is extensive and the presence of acids as a causative factor is suspected, the regular use of alkaline mouth washes is helpful in preventing recurrence. They may also be used as a part of the treatment. A mouth wash that most nearly meets the requirements is lime-water. As its preparation and use have been outlined in the chapter on Postoperative Care, they will not be repeated here. Lime-water is simply a saturated solution of calcium oxide and can be made up at a trifling cost by any patient. It can also be obtained of the druggist, but in that event it is desirable that it be made from the commercial instead of the chemically pure calcium oxide, as the latter is said to be less efficient in treatment. Why this should be so is uncertain, and where it is put up by the druggist the difference is probably so slight that the chemically pure calcium oxide can be used if the commercial is not available. The chief objection to its use is its not altogether pleasant taste. This can be modified by adding a little saccharin or other flavoring material as indicated in the prescription given in the chapter on Postoperative Care. It is undoubtedly the most scientific and effective mouth wash available for the neutralization of mouth acids whether in cases of dental caries or exposed and hypersensitive root surfaces. In most cases in which it has been made up by the patient it is desirable to begin its use by diluting it somewhat, but the patient should be encouraged to eventually use it full strength.

Another mouth wash designed to meet the same require-

ments and one which can be put up by any druggist is the following:

Calcium oxide	75 grains
Menthol	7 grains
Saccharin	6 grains
Shaker salt	½ pound

Sig: A quarter of teaspoonful in a half glass of warm water and use as a mouth wash several times daily and always just before retiring.

In compounding it the ingredients should be thoroughly triturated.

THERMAL SHOCK

The *second type* of sensitive teeth due to gum recession and loss of tooth substance is one in which the dominant symptom is sensitiveness to changes in temperature. In these cases an effort should be made to ascertain how much of this is due to sensitivity of tooth surface, and how much to actual disturbance of the pulp itself. A differential diagnosis can usually be made by noting the degree of sensitivity and the amount of the exposure including loss of tooth substance. In those cases in which the exposure is considerable and the surface highly sensitive, the symptoms of thermal shock which may be present under these conditions are probably due to the exposed and highly sensitive root surface, rather than to disease of the pulp, and can be controlled by the application of some of the desensitizing preparations above referred to. There is, however, a type in which there is little or no surface sensitivity, and where the chief or only complaint is pain from change of temperature. This pain is usually induced by the introduction into the mouth of something hot or cold, though it may be brought about by the patient's being out in the open air. This second type is potentially more serious than the first as it indicates a mild hyperemia of the pulp, which may,

if not controlled, result in its death. This hyperemia is of two kinds: arterial and venous. When the pulp of a tooth is shocked by changes in temperature, the vasodilators cause dilatation of the capillaries of the pulp which, by their expansion, bring about pressure on the nerves and so cause pain. Immediately the vasoconstrictors react, the pressure is relieved and the pain ceases. If, however, these shocks are long continued or are severe, the walls of the capillaries are injured and are no longer able to contract under the influence of the vasoconstrictors. The result is a passive or venous hyperemia. A differential diagnosis can usually be made by the length and severity of the pain. When the capillary walls are normal they contract promptly and pain ceases almost instantly. This is the case in arterial hyperemia. In venous hyperemia, the paroxysm is more prolonged and the prognosis correspondingly uncertain. Fortunately there are few cases of venous hyperemia caused by gingival recession, though arterial hyperemia, usually of a mild type, is common. Occasionally it will take the form of a chronic facial neuralgia, as is illustrated by the following case. The patient, a middle-aged woman, when first seen had been in bed for five weeks. She gave a history of facial neuralgia of several years' standing, which during the warmer seasons would practically cease altogether but was more or less continuous during cold weather. Several physicians and dentists had been consulted. As no relief had been obtained it was decided to remove an impacted mandibular third molar, in the belief that it might be the cause of trouble, which was done by a general surgeon and under a general anesthetic. Infection and suppuration subsequently occurred making drainage by an external incision necessary. Meanwhile the neuralgic pains continued unabated and it was while the patient was convalescing from this operation

that she was first seen by the author. A bedside examination revealed the presence of all the teeth except the third molars, and all free from caries or periodontoclasia. There was, however, a very marked recession of the atrophic and abrasive types, involving more or less all of her teeth. As they were exceedingly sensitive to any change of temperature, a diagnosis of arterial hyperemia was made. The teeth were protected by a napkin and the exposed root surfaces washed off with warm alcohol and dried. Each receded area was then painted with a nonconducting varnish to be described later, which was reapplied as found necessary until the patient was able to come to the office. The relief was almost immediate. Within twenty-four hours all pain had ceased and there was no recurrence as long as they were protected. Subsequently, two of the teeth were devitalized. Up to the time of her death, fourteen years later, there were but two mild relapses, both of which were promptly controlled by another application of the nonconducting varnish. By temporarily protecting them in this way the pulps were relieved of irritation to the extent that they subsequently entirely recovered without resort to any other treatment except in the two teeth above referred to. This, of course, was an exaggerated case, but shows how great a disturbance of the pulps may occur from nothing more serious than gingival recession and root exposure.

TREATMENT

As in these cases sensitiveness to thermal shock is due to the recession of the gums and exposure of the dentine and cementum, their logical treatment is to protect these areas against temperature change by some covering. Having protected the teeth against moisture, this is most satisfactorily accomplished by washing off the exposed surface

with warm alcohol to remove any gelatinous plaques that may be present, and, having carefully dried the surface with warm air, by flowing over it a thin mix of oxyphosphate of zinc. If the tooth be thoroughly dried, the adhesiveness of the cement will usually be sufficient to hold it in place long enough for the pulp to recover. Generally a few weeks will suffice. If it is brushed off as it may be it can be replaced. Unless decay be present or a considerable portion of the tooth and root has been destroyed by abrasion, there are few cases that justify cutting into the tooth for the insertion of a filling.

A NONCONDUCTING VARNISH

The nonconductivity of these fillings can be greatly increased by first covering the exposed surface with a nonconducting cavity varnish such as that referred to above. One that is almost ideal in this respect is made as follows:

Gum damar	1 dram
Commercial zinc oxide C. P.	1 dram
Canada balsam	1 drop

The gum damar should be liquefied by dry heat, after which the other two ingredients are added and incorporated in the mixture by a thorough stirring. It is then poured into a small jar and allowed to cool, when it takes the form of a white resinous substance. To use it, saturate a pledget of cotton in chloroform and rub it on the surface of the varnish until saturated. Having washed off the exposed surface of the tooth with warm alcohol and carefully dried it with warm air it is then applied, covering the entire surface. To hasten the evaporation of the chloroform, use the chip blower or compressed air. When thoroughly dry, which will take a few minutes only, oxyphosphate of zinc may then be flowed over it. As a nonconductor, it is little less than perfect. Teeth that are so sensitive

that any change in temperature causes pain, can in many instances be completely controlled by its application. Because it will be brushed off by the toothbrush, it is usually desirable in these cases to cover it with cement. As it is very adhesive, it will, if applied to a dry surface, often remain intact for a week or more before being entirely brushed off, which in some cases may be sufficient to insure the tooth against further shock. As a nonconducting lining under fillings it has no equal. It should always be applied to a *dry* surface, the chloroform evaporated and the edges of the cavity cleaned of the varnish before inserting the filling. In deep cavities in which a lining of cement is necessary to protect the pulp, it can at the same time be protected against the irritating effects of the phosphoric acid by first painting the surface of the cavity with the varnish. For the same reason it is useful as a pulp protector under silicate cement fillings. It is a preparation that anyone can make in a few minutes, or it can be put up by the druggist at a trifling cost, having first instructed him in the way in which it should be prepared.

SUBGINGIVAL SENSITIVE AREAS

Another type of case is one in which the sensitiveness is subgingival, such as is occasionally met with in cases of periodontoclasia. In this type there is usually no symptom until the instrument is brought to bear on the root surface in subgingival curettage. Occasionally, though not often, cases will occur in which it is impossible to operate without an anesthetic because of this subgingival sensitiveness. Where this is marked and where it involves more or less all of the teeth, one should suspect diabetes and have a urinary analysis made. In a case of periodontoclasia recently seen by the author, the root surfaces were so sensitive that it was impossible to touch them with an instrument without

causing exquisite pain. A urinary analysis revealed the presence of six per cent of sugar and a blood examination 400 milligrams per 100 c.c.

Conductive anesthesia is the only way in which to treat these cases. If an individual tooth only is involved, an infiltration that will anesthetize the pulp will serve the purpose. There is no other way in which these cases can be controlled. Following treatment, there are usually no symptoms save a possible increased sensitiveness to temperature changes. This is to a certain extent true of all cases treated by subgingival curettage. The patient is warned to expect this and told that it is only temporary. Usually these symptoms disappear in a few weeks. They are due to the planing operation on the root surface and tend to disappear as healing in the tissues progresses, though where there is considerable recession there may always be some reaction to marked changes in temperature.

MISCELLANEOUS CASES

Teeth on which gum resection has been practiced are always more or less sensitive and may require protection during the period of convalescence. A preparation known as "Wonderpak" has been designed to meet this situation.[3] It is a slow-setting medicated cement, which is packed around and between teeth that have been operated upon, and allowed to remain until healing has taken place. Usually a couple of weeks is all that is necessary.

When the bifurcations between the roots are exposed, either from gum recession or resection, and where it is desirable that something be done to protect them against thermal shock and the accumulation of food in these spaces, they can be filled very satisfactorily with oxyphosphate of copper cement. It is more or less permanently antiseptic and the soft tissues are not irritated by it. Its color pro-

hibits its use where it will be seen. By the use of some one or all of these agents, there are few cases of hypersensitiveness of exposed root surfaces that cannot be controlled.

REFERENCES

[1] Bodecker, C. F., 1927, "The Most Sensitive Areas of the Teeth and their Operative Treatment," *Journal American Dental Association,* vol. 14, p. 1643.

[2] Payne, J. L., 1928, "A Paste for Obtunding Sensitive Dentine," *Dental Cosmos,* vol. 70, p. 1231.

[3] Ward, A. W., 1928, "The Surgical Eradication of Pyorrhea," *Journal American Dental Association,* vol. 15, p. 2155.

CHAPTER XIX

SPLINTING TEETH

Splints have a very limited place in the practice of periodontia. The tooth that is so loose that it cannot be rendered healthy and useful by treatment should be removed. Unfortunately this attitude toward the problem of loose teeth does not always prevail and attempts are sometimes made to retain teeth that should be extracted. Frequently this is not the dentist's fault. The patient is usually so desirous of saving his teeth that he will not consent to extraction even though advised to do so, especially if the teeth are comfortable. In such cases the dentist's responsibility ceases with the advice given, note of which should be made a part of the patient's record. The tendency of all teeth loosened by periodontoclasia is to become noticeably firmer following treatment, though in some cases they may never become entirely firm. Indeed, in many instances they do not, but if health can be reëstablished in the tissues surrounding them, they will often continue to function indefinitely with almost perfect satisfaction to the patient. It is this type of tooth that has to be carefully guarded against excessive occlusal stress by judicious grinding. Because of the weakened condition of such teeth through the partial loss of their supporting tissues, they should be ground sufficiently to compensate for the changed conditions. This is made necessary in order to lighten the load rather than because of any abnormality in occlusion.

FACTORS CAUSING TEETH TO LOOSEN

The loosening of teeth from periodontoclasia is due to four possible factors: (*a*) bone loss; (*b*) gum recession and inflammation; (*c*) thickening of the pericementum, and in many instances to (*d*) a certain amount of rarefaction in the remaining alveolar support. In the consideration of these cases this last factor is frequently overlooked. Periodontoclasia not only destroys the alveolar bone, but in the process of destruction weakens to some extent that which remains by withdrawing its mineral content. This condition of semi-rarefaction will be evident in the radiographic examination of many cases as will be the increased density which so frequently follows treatment.

Correct treatment should improve all these factors. In many cases there will be some reformation of bone; occasionally it will be more or less complete, though this is the exception and not the rule. It is not to be depended upon. The most marked change in this respect takes place in the remaining alveolar process in which new lime salts are deposited, increasing thereby its density and contributing to increased tooth support. Under the influence of treatment, the diseased and thickened pericementum also undergoes regeneration which further adds to the process of tooth rigidity. But more important than all of these in its contribution to the stabilization of loosened teeth, when there is little or no re-formation of bone, is the restoration of the gum tissues to health. When involved in periodontoclasia they are usually inflamed and stand away from the roots of the teeth in such a way as to afford almost no support to the teeth. Following subgingival curettage, all this is changed. The inflamed and relaxed tissues at once take on the attributes of health. They become firm and hard, closing the spaces created by the pockets with their content of calculary deposits and septic materials, hugging the roots

firmly and in many instances reattaching themselves to the root surfaces. This is another reason why the gums should never be cut away except as a last resort, for in every case they provide one of the important factors in stabilizing teeth loosened by periodontoclasia.

For the foregoing reasons it will be seen that splints can have but a very limited place in periodontia. In most cases in which the disease is so advanced that the teeth cannot be held sufficiently firm by their natural support, they should be extracted. Teeth that can be slightly rotated or elevated or depressed in their sockets are usually hopeless and should be removed. There are cases, however, in which some form of splint may be used to advantage, though such cases are the exception and not the rule.

TYPES OF SPLINTS

One of these is a temporary splint to be used to support certain loose teeth during the period of convalescence. It not infrequently happens that the lower incisors, for example, are very loose and are made temporarily looser by curettement, making it desirable that they be firmly held in place until the surrounding tissues have had an opportunity to recover. These can be held perfectly and the patient made entirely comfortable by ligating them to firm teeth on either side. Linen thread, Japanese grass line, or the fine-braided silk ligature used in orthodontia may be employed after it has been carefully waxed. It should always be of small size and care used in adjusting it so as not to displace any of the teeth. Passing it around the firm tooth on one side, it should be tied securely with a surgeon's knot, after which each tooth in turn is tied till it is fastened to a firmly held tooth on the opposite side of the group of loose teeth. In each instance the knot is tied in the proximal space. When this is wide two or more knots

should be made, to avoid traction on the loose teeth. It should be placed far enough above the gingivæ to prevent impingement. It is desirable to change it at fairly frequent intervals, and to remove it altogether just as soon as the tissues themselves provide the necessary support. How often it should be changed and when it can be removed altogether will, of course, depend upon the conditions present. A few weeks will usually suffice and two or three changes of the ligature be all that will be necessary. This type of splint is not applicable to the molars but can be used anywhere else in the mouth, though it is best employed in stabilizing the lower incisors. As a temporary support for certain teeth it serves a useful purpose. Wire may in some instances be preferable to thread.[2] It has the advantage of being nonabsorbent.

PERMANENT SPLINTS

There are very few places in which permanent splints are indicated and it is the author's belief that they are never applicable to the support of molar teeth. A molar so loosened by disease that it cannot be made to function satisfactorily without artificial support should be extracted. It is in a region of the mouth subjected to the greatest occlusal stress, and where it is naturally most difficult to keep teeth clean. Moreover, it is well-nigh impossible to adapt any form of splint to molar teeth that will successfully immobilize them and which will at the same time permit of proper hygiene. For these reasons, splints on molars are with very few exceptions contraindicated. Indeed, the use of all fixed splints is practically limited to the incisors. A case from practice will illustrate their most logical application: Two lower central incisors from around which more than three-quarters of the bone had been destroyed had been further complicated by the loss

of nearly as much of the supporting gum tissue. The teeth on either side were firm. There was no difficulty in establishing health in the surrounding tissues, but they were insufficient to hold the teeth firmly. They were loose enough to permit of considerable anterioposterior movement which, unless controlled, would lead to their eventual loss. As the teeth were otherwise healthy it was decided to employ a permanent splint rather than extract them and subject the patient to a partial denture or any form of bridge. This was done by the use of a delicate pinledge splint.[1] Attachments were made to each of the four teeth just lingual to the incisal edge and soldered together at the contact points. When set, the teeth were firmly held. They could be easily cleaned by the use of brush and toothpicks and the patient was spared the inconvenience of artificial teeth. (Fig. 56.)

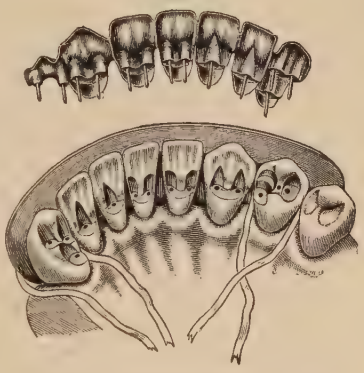


FIG. 56.—Permanent splint as devised by Burgess.

They can, of course, be used in other locations, but it is in the incisor region that they serve their most useful purpose. Under no circumstances does their application justify the devitalization of teeth as has sometimes been done in the past. When used with discrimination they have a place in periodontia, since they permit of the saving of teeth that might otherwise be lost.

REFERENCES

[1] BURGESS, J. K., 1915, "Stabilizers for Loose Teeth," *Dental Cosmos*, vol. 57, p. 1344.

[2] PRIEST, C. A., 1914, "Temporary Retention of Loose Teeth," *Dental Cosmos*, vol. 56, p. 316.

CHAPTER XX

TOBACCO SMOKING: ITS RELATION TO THE SOFT TISSUES OF THE MOUTH

It is a matter of more or less common observation among those who have made a study of the subject, that the smoking of tobacco may be prejudicial to the health of the oral tissues. Inquiry in such cases leads one to believe that the resultant injury is dependent upon three factors: (*a*) the type of tobacco smoked; (*b*) the amount smoked, and (*c*) the individual's susceptibility. There can be no doubt that there is a marked difference in the matter of susceptibility. Some individuals can smoke to excess without any appreciable influence upon the oral mucosa, while others who are moderate smokers may exhibit striking effects. Other things being equal, however, the effect upon the mouth will be in direct ratio to the amount of tobacco smoked. This seems to be a greater factor than the kind of tobacco, whether it be that used in pipe, cigar, or cigarette.

Practically every dentist has seen cases of leukoplakia due to excessive tobacco smoking. Less often perhaps is note made of the mouth in which the oral mucous membrane is markedly pale in color and in some instances having a lifeless, parboiled appearance, especially noticeable on the hard palate. Occasionally this will take on a pebbly look in which there is some pigmentation. This condition probably represents, in some instances, one of the early steps in leukoplakia, though in this stage of development it can hardly be diagnosed as such.

These conditions are brought about by the irritating effect of smoking, and in some instances there can be no doubt that they unfavorably influence prognosis in the treatment of periodontal diseases and act as predisposing factors in etiology.

COMPOSITION OF TOBACCO

Tobacco contains a large amount of salts, consisting of sulphates, nitrates, chlorides, phosphates and malates of potassium, calcium and ammonium. Other constituents are albumin, resin, gum, tannin, citric acid, nicotianin and nicotine. The poisonous principle in tobacco is chiefly nicotine, though collodine, pyridine and picoline are factors to be reckoned with.

The smoke of tobacco is even more complex in its chemical composition than is the tobacco itself. When burned, it undergoes a destructive distillation, yielding many decomposition products, just as coal, when heated, yields a multitude of dyes, medicines, disinfectants, etc. Hydrocyanic acid, ammonia, formaldehyde, and hydrogen sulphide are among the constituents of tobacco smoke. Nicotine with its decomposition products, especially pyridine, is the stain-producing substance in tobacco so often seen on the teeth of smokers. It is these substances that take the form of a black tarry mixture that forms in the stem of the pipe and gives rise to its offensive odor.

The quantitative composition of tobacco smoke naturally varies with the kind of tobacco used, its form and burning quality. As revealed by chemical analysis, 100 grams of tobacco smoke contain the following ingredients:

Nicotine	1.16 grams
Ammonia	0.36 gram
Pyridine	0.15 gram
Carbon monoxide	4124 c.c.

The smoke from one cigarette as shown by chemical examination is composed of the following ingredients:

Nicotine	5	milligrams
Ammonia	1.5	milligrams
Pyridine	0.7	milligram
Hydrocyanic acid	0.03	milligram
Carbon monoxide	18	c.c.

ITS INFLUENCE UPON PERIODONTAL CIRCULATION

The long-continued application of nicotine and its decomposition products to the soft tissues of the mouth has the effect of restricting their blood supply. Observation made in the experimental production of arteriosclerosis has revealed the fact that nicotine "acts directly on the muscular middle coats of arteries." [1] Its effect is that of a mild angiostenotic. Its influence upon the capillaries of the oral mucous membranes is one of contraction; the capillary walls are made narrower, with the natural result that the tissues receive less blood and are therefore less well nourished.[3] It is as though one who had been in the habit of watering his garden with an inch hose should substitute for it a half-inch hose. The effect would be that the garden would receive only one-half as much water as formerly and by so much would be injured.

This is an important factor to be considered in the relation of tobacco smoke to periodontal diseases. As was pointed out in the chapters on etiology and treatment, undernourishment of the tissues of the periodontium is a very considerable factor in their susceptibility to disease. Lack of vigorous use of the teeth and jaws in mastication was observed to be one of the explanations of the undernourishment of these tissues. When to this almost universal condition of functional inactivity is added the restrictive influences upon circulation of tobacco smoking, it requires no stretch of the imagination to realize that smok-

ing is a factor to be considered in the etiology and treatment of all periodontal diseases when seen in the mouths of smokers. As already indicated, some individuals are more susceptible to its influence than others, but while this is true, it is equally true that tobacco smoking adversely affects the prognosis and treatment in all cases unless used in extreme moderation.

SMOKE AS A GERMICIDE

Tobacco smoke is mildly disinfectant, though its germicidal influence is more or less transitory.[2] To what constituent or constituents of tobacco smoke this antibacterial effect is due is a matter of uncertainty. Some believe it to be due to nicotine, but this has been disputed by others. The presence in tobacco smoke of formaldehyde may explain it, as it may also explain some of its irritating effects upon the oral mucous membrane. The disinfectant influence of smoke, however, is of such a temporary character as to be of little or no value in the inhibition of dental caries, and of no value whatever in the prevention of periodontal diseases. Its irritating effect far exceeds any value which it might possess as a germicide. In a report on the germicidal influence of tobacco smoke, Appleton, who has done some excellent work in this field, states: "Smoking one cigarette reduces the number of bacteria removable by rinsing the mouth by about 40 per cent. Thirty minutes after smoking, this number is still 14 per cent below the initial number and 26 per cent below the corresponding number of the control group. Sixty minutes after smoking there appears to be no difference between the smoker and the control groups." [2] He also states that "Trillat demonstrated formic aldehyde (formaldehyde) in the smoke from cigars, cigarettes and from pipe tobacco." [2]

LEUKOPLAKIA: ITS PATHOLOGY

While leukoplakia can hardly be classed as a periodontal disease, affecting as it may any of the soft tissues of the mouth including the tongue, it is, nevertheless, one which should receive serious consideration from the dentist. It is usually represented as a grayish-white, striated patch, located on some of the oral mucous membranes. It may appear on the gums, palate, tongue or on the inner surfaces of the cheeks or lips. As the disease progresses, the involved tissues take on a hard and leathery appearance due to the thickening of the epithelial covering. These patches are most apt to occur at the angles of the lips, behind the molars and along the borders of the tongue. In advanced cases they show a tendency to crack, resulting in fissures or ulcerations that are more or less painful.

ETIOLOGY

The most common cause of leukoplakia is tobacco smoking. Syphilis has been regarded as a frequent etiologic factor and at one time was held to be pathognomonic, but more recently investigators incline to the belief that it is not commonly a direct cause. Irritation from any cause, such as broken-down carious teeth, badly adjusted restorative dental operations, biting the inner surfaces of the cheeks or lips, highly seasoned foods, alcohol, etc., all predispose to the development of leukoplakia. But by far the most common cause is the excessive use of tobacco. Whatever may be the active exciting factor, it is the irritation which it produces on the oral tissues that in many instances causes the development of leukoplakia.

TREATMENT

Inasmuch as leukoplakia must be regarded as precancerous lesion, the treatment is a matter of no little

importance. This consists in the removal of all irritants of whatever nature and the discontinuance of tobacco in all its forms. Attention should be given to the establishment in the mouth of a high standard of hygiene, which should be maintained by careful personal care and periodic prophylactic treatments. Cauterization of the leukomatous patches or the use of escharotic drugs should be avoided, as they tend to increase rather than diminish the irritation. Surgical removal of the affected mucous membrane may, in rare instances, be desirable. This is in cases where there are deep fissures, bleeding, pain, etc., in which event a specialist should be consulted.

As a rule, the only treatment required is the discontinuance of tobacco and the maintenance of good hygiene. Under these conditions, the white and irritated patches usually completely disappear in the course of a year or two. In a case seen by the author in November, 1928, in which the entire hard palate was involved, presenting a grayish roughened appearance, more than three-quarters of the surface had entirely cleared up six months later. The remaining one-quarter had improved though the surface was still noticeably elevated above the surrounding tissues, and was somewhat pebbly in appearance. It is probably only a question of time when the remaining patches will also disappear. The only treatment in this case was the discontinuance of smoking which had been at the rate of ten or twelve cigars daily for a period of more than twenty years.

SUMMARY

There can be no doubt that the smoking of tobacco adversely affects the periodontal tissues, in some instances predisposing them to disease by restricting their blood supply and unfavorably influencing prognosis. How large a

factor it may be in a given case it is impossible for anyone to determine. In those cases of periodontoclasia in which there is marked evidence of excessive tobacco smoking, the patient's attention should be called to the condition that exists and he should be advised to smoke less. Where there is noticeable pallor of the oral mucosa, with that dry and half-cooked appearance of the gums so characteristic of some of these cases, the prognosis is distinctly unfavorable unless the patient is willing to discontinue smoking altogether or at least greatly reduce the amount. The author has advised patients who were unwilling to modify their smoking habits that he did not believe it was worth their while to undertake treatment under the prevailing conditions. Such cases of periodontoclasia can undoubtedly be helped, but the results can never be wholly satisfactory. Where their treatment is undertaken, it should be with the understanding that the operator cannot be held entirely responsible for the outcome. All patients who smoke should be advised that it unfavorably influences prognosis in the treatment of all periodontal diseases, and that in cases of leukoplakia and Vincent's infection, they should forego smoking altogether until after all symptoms have disappeared. Moderate smoking will not seriously interfere with the treatment of the average case of periodontoclasia, though it is probable that even such smoking adversely affects both the periodontal tissues and the hygiene of the mouth.

REFERENCES

[1] Adami, J. Geo., 1911, "Principles of Pathology," pp. 6, 7, 305, Lea & Febiger, Philadelphia, Pa.

[2] Appleton, J. L. T., and Lehmer, E., 1928, "The Effect of Cigarette Smoking on the Number of Bacteria Removable in Mouth Rinsing," *Dental Cosmos,* vol. 70, pp. 1111, 1113, 1121.

[3] Bunting, R. W., 1929, "Oral Pathology," p. 324, Lea & Febiger, Philadelphia, Pa.

[4] Wolf, G., 1924, "Has Tobacco Smoke a Disinfectant Action?" Abstract *Dental Cosmos,* vol. 66, p. 1381.

CHAPTER XXI

PERIODONTIA AS A SPECIALTY

Periodontia is so vitally a part of the general practice of dentistry, and so fundamental to its success that it cannot be logically separated from it. If periodontia is that branch of dentistry concerned with the diagnosis, prevention and treatment of diseases affecting the supporting tissues of the teeth, as it has been defined as being, all will agree that this should be the concern of the entire dental profession. Care for the health of these tissues cannot be relegated to a mere handful of specialists; it is, or should be, the business of every dentist. If the supporting tissues of the teeth are not maintained in health; if in daily practice, the family dentist is so intent on repairative treatment that he has no time for periodontia, dentistry is to that extent a failure, and the public by so much the losers.

CASES FOR THE GENERAL PRACTITIONER

If the general practitioner will keep in mind that there are no diseases occurring in the mouth more easily prevented than those involving the periodontium, and that in their incipiency all are easily cured, he will realize that there can be no excuse for neglecting them. His failure in this respect is due in large measure to the fact that in the past he has recognized the existence in the periodontium of but one disease, and then only after it had reached an advanced stage. This disease he called "pyorrhea." Without understanding its etiology and with instruments wholly unsuited to its treatment, he has made the attempt and

failed, as it was inevitable that he would do under such conditions. The natural result was the belief that it could not be cured. And even at present, patients are being told by their dentists that "pyorrhea is incurable." The mistake has been made in allowing such cases to reach an advanced stage before beginning treatment, at which stage treatment is always difficult and the outcome not always certain. If treatment is long enough delayed, every case will reach an incurable stage and justify the statement, when it has progressed to that stage, that "pyorrhea is incurable."

Another explanation for this neglect has been the mystery with which periodontoclasia has always been regarded. This has, no doubt, been increased in more recent years by the addition to its nomenclature of many new and unfamiliar terms, and by dividing and subdividing the few diseases of the periodontium until they appear an appalling list. This has further complicated the situation.

The attitude of the family dentist toward this whole problem, as indeed toward every problem in dentistry, should be that of prevention. The gingival tissues of every patient presenting for treatment should be carefully examined. Nothing should be taken for granted. The subtle and unobtrusive way in which lesions of the periodontium have their beginnings, and the frequency with which they occur, make it doubly important that no chances be taken. The gingival crevice of every tooth should be explored and its contents removed each time the patient presents for treatment. The teeth should be cleaned in a most thorough and painstaking way. Some preparation as a disclosing stain should be used and allowed to bathe the teeth and gums. Iodine preparations of proper strength, as already indicated, are admirably suited to meet this requirement.

In those cases in which disease is present in its early manifestations, treatment is not difficult and the prognosis is good. The skill necessary for taking care of all such cases is easily acquired once the principles have been learned. With practice, one's resources are increased to the point where more advanced cases can be successfully treated.

CASES FOR THE SPECIALIST

Into every practice, however, there will come the advanced case, in which some teeth have already been lost and others are so nearly hopeless that saving them seems doubtful. It is such cases that require great skill and wide experience in treatment. One such came into the hands of the author in 1903. He was then a professional infant with no knowledge of periodontia and no experience whatever in its practice. The patient, then thirty-six years of age, had been told by her dentist in an adjoining city that her case was hopeless. The author agreed with him that that was probably true, but was so impressed with the desirability of saving what had been a beautiful set of teeth that his interest was aroused. In the spirit in which "fools rush in where angels fear to tread" (and sometimes win), he attempted its treatment, and from that day to the present his interest in periodontia has grown day by day and is still growing. It was his baptism into periodontia. The patient, now over sixty years of age, is still under his care and has most of her teeth. Had he known enough about periodontia in 1903 to have given her the care that she required, she need not have lost any.

These are the cases that require the services of the specialist or at least some one with a wide experience in periodontia. And specialists there will probably always be, who because of an aptitude for the work will engage in perio-

dontia as a specialty. Devoting all their time to it, they will naturally acquire a larger degree of skill. The dentists who are now engaged in periodontia as a specialty of dentistry were all at one time general practitioners, who made the practice of periodontia a part of their daily work. As they became more proficient in its practice, the demands upon their time became such that they were gradually forced out of general practice. And so they became specialists by necessity. This is as it should be. No one should go directly from the dental school into periodontia. He needs the background of general practice. Only those who have had it and who have developed skill in periodontia should take it up as a specialty. It is to such men that the general practitioner can refer his more difficult cases, and obtain for them the treatment which he, with a more limited experience, may feel he cannot give them.

It is at this point that mistakes are most apt to be made. The difficulty lies in determining which cases shall be treated by the general practitioner and which shall be referred to the specialist. There can, of course, be no fixed rule by which a decision can be made, as that will depend upon the nature of the case in hand and the resources of the family dentist in the treatment of such cases. The mistake is usually made in attempting too much. No attempt at deciding this important matter should ever be made until after a complete radiographic examination has been made. The difficulties will usually be found to be in direct ratio to the depth of the pockets and the total number of teeth that have been lost. It is generally the part of wisdom to err on the side of safety by referring to the specialist all cases about which there is any doubt. If for any reason the patient is unable to follow such advice, the responsibility is his and not the dentist's; he at least will have done his duty.

SUMMARY

The general practitioner then should qualify himself to diagnose all periodontal lesions and to give such treatment —preventive and curative—as lies within the limits of his capacity. In so doing he should be able to care for the periodontal needs of most of his patients. Those that remain can be sent to the specialist. By so doing, he can greatly increase his usefulness to his patients and render them a service which in many instances is urgently needed. Moreover, patients are more and more realizing their need of such service, and with increasing numbers are demanding it of the family dentist. If for any reason he feels disqualified to undertake the treatment of a given case, the patient's confidence is in no way abated by being referred to a specialist. His confidence is destroyed only when he feels that he has been neglected.

REFERENCES

[1] Schott, C. H., 1925, "The Dental Hygienist and Dental Assistant in Periodontia Practice," *Journal American Dental Association,* vol. 12, p. 5.

[2] Merritt, A. H., 1925, "Building a Periodontia Practice Without camouflage," *Journal American Dental Association,* vol. 12, p. 3.

[3] Rich, Celia, 1925, "Periodontia Service for the Poor," *Journal American Dental Association,* vol. 12, p. 7.

[4] Stillman, P. R., 1925, "The Relation of the Periodontist to Other Members of the Dental and Medical Profession," *Journal American Dental Association,* vol. 12, p. 9.

CHAPTER XXII

PERIODONTIA: ITS EDUCATIONAL AND ECONOMIC ASPECTS

The author was once asked, "How can a dentist practice periodontia in a community in which every other dentist cleans teeth for a dollar and a half?" The question represents a problem with which many are confronted and especially those practicing in smaller towns and cities. It is a problem with two angles—one educational and the other economic.

In large letters on one of the beautiful panels that adorn the walls of the Congressional Library in Washington are these words: "Ignorance is the curse of God. Knowledge the wing wherewith we fly to Heaven." To a large extent, ignorance is at the bottom of the problem contained in the above question and knowledge is its solution. The problem is primarily that of the dental profession, whose duty it is to educate the public in all matters pertaining to dentistry, a duty that is being fulfilled by many dental organizations throughout the United States, notably by the American Dental Association and the American Academy of Periodontology. Both of these organizations stand ready to coöperate in any effort that is being made to advance the public's knowledge of dentistry in all its different aspects, with the emphasis placed where it should be, on prevention. Through its Publicity Committee, the American Academy of Periodontology has prepared a long series of articles, which have been designed for use in the public press and which cover every aspect of dentistry. These

are available to anyone and have met with widespread approval and a most gratifying response wherever they have been used, which includes practically every state in this country and more than a dozen foreign countries, among them Australia, New Zealand, Greece and South Africa.

THE EDUCATION OF DENTIST AND PUBLIC

The advice given to the above-mentioned inquirer was that he begin a campaign of education in his local dental society, a task that is neither difficult nor impossible. A little perseverance and enthusiasm tempered by good judgment will do much in such situations. What is needed is some one to take the lead. It is not difficult nor is it expensive to get talent from outside to assist. The time was when it was not easy to interest dentists in periodontia, but that day is past. More and more are they realizing its importance. All that is needed is to present it to them, freed from the "isms" which for so long have blocked the wheels of progress.

In addition the inquirer was advised to "clean teeth" so well that it could and would be said of him in his community that nobody else cleaned teeth as he did. Also that he make it a part of his daily service to his patients to educate them in what is meant by a clean mouth; its relative freedom from disease, pain and tooth loss, and that this type of service costs less than the repairative kind. In this way a certain number would learn at first hand a most important lesson, and this would act as a leaven in that community which, though it might not "leaven the whole lump," would be a step in the right direction. Such face-to-face instruction, which is the most helpful, supplemented by the intelligent use of educational bulletins such as those already referred to, should do much toward

solving one of the problems involved in the above question.

THE ECONOMIC SIDE OF PERIODONTIA

But it has also an economic angle. The author is frequently told that patients will not pay for periodontic treatment, that to them all such treatment and especially so if it be of a preventive nature, is "just a matter of cleaning teeth" and that patients will not pay to have their teeth cleaned, at least will not pay adequate fees. And in every community it would seem that there are those who are willing to "clean teeth for a dollar and a half." To a certain extent this is true, which may explain many surgical operations in periodontia since they cannot be confused with "cleaning teeth," hence larger fees. The truth of the matter is that the public has been in the habit of buying fillings, crowns, bridges, etc., of the dentist, instead of buying service. It has come to think of dentistry in these terms. It has been educated into placing its reliance upon this kind of dentistry. This is wrong. The dental profession must itself place the emphasis where it belongs, by the practice of prevention as a part of its daily work. Patients need to be told that it is entirely possible with their coöperation, in the light of present knowledge, to prevent not less than 75 per cent of all dental maladies. Already this is being realized by the more intelligent among them, who are demanding that repairative dentistry give way to preventive dentistry. Again it will be seen that it is a matter of education and that the dentist must be the teacher.

It is also true that there are people who cannot pay for any kind of health service or can pay but little. Every member of the dental profession must share in the responsibility of doing what he can to meet this situation. It is

his duty to do this, as it is his duty to see to it that those of his patients who can afford to do so pay his fees. These should always be reasonable and never exorbitant. The Golden Rule is the best guide by which to regulate them.

PERIODONTIC TREATMENT NOT EXPENSIVE

Periodontic treatment even in advanced cases need not be expensive and in preventive cases never is. Occasionally one hears of the large fees some one is reported to have charged, and the impression goes abroad that "pyorrhea treatment" is costly. It is not. There are few cases that require more than a few hours of treatment and the cost per hour is always less than that of restorative treatment where the cost of supplies has to be considered. And the cost of treatment can always be reduced by the application of efficient methods. The individual who by such methods can in one hour do what another requires two hours to do, has, other things being equal, cut the cost of that operation in half no matter what his fees may be. Indeed, he may charge a fee per hour twice that of his colleague and at the same time be the less expensive of the two, for there is the cost of time, transportation and wear and tear to be considered. Unfortunately, if he quote fees based on hourly charges, he will be placed in the position of appearing to be twice as expensive, which is only one of the reasons why such fees should never be quoted. In furnishing the patient with an estimate of the cost of a given case, one should have in mind the probable amount of time required in treatment, the value of one's time, which is largely governed by the demand made upon it, and then quote a fee for the completed operation. Many patients uneducated in such matters are in the habit of thinking of dental fees in terms of hourly charges, a situation for which the dental profession is largely responsible. If all were of equal skill,

and could accomplish the same results in the same time, this might be a satisfactory way of estimating fees, but since this is not so, and in the very nature of things never can be, one should learn through the application of efficient methods to render the maximum of service in the minimum of time, and then charge for the service rendered, not for the time consumed. Only by such methods can economic justice be done to all concerned. When applied to periodontia, the cost of treatment will always be found to be less than that of neglect, involving as it does the eventual loss of the teeth and the expense of replacement. It will also have a greater value. These facts should be explained to the patient, who naturally cannot be expected to understand them, in order that he may see for himself that it is not the cost of treatment that is expensive, but the lack of it.

FEES IN PERIODONTIA

Nothing is more unpleasant in the practice of periodontia than misunderstandings regarding fees. For this reason, one should take all possible precautions to avoid them, inasmuch as most of them can be avoided when proper measures are taken.

One of the most effective ways in which to prevent such misunderstandings is to have the confidence of the patient. If he feels that he can trust the person to whom he applies for treatment, there will be little trouble about fees, providing, of course, that his confidence is not misplaced. The ability to inspire confidence in another naturally varies with different individuals. Some possess it in large measure, while others are less fortunate. Personality, frankness and tact are all important elements in creating it. But more essential than all these as they apply to the practice of dentistry (for periodontia is only a part of dentistry), is a thorough knowledge of one's subject and the ability

to do with the hands that for which one's services are sought.

When thus equipped, one unconsciously inspires confidence in the patient, who intuitively feels that he is in the hands of one whom he can trust to not only do what is required of him, but to charge a fee that will be entirely just. Mastery of one's subject with the ability to do one's work well are the stepping-stones to confidence, no matter what the work may be.

When to these are joined honesty of purpose and a sincere desire to put service above fees, there will be few misunderstandings regarding the cost of periodontic treatment on the part of those who appreciate the importance of such treatment, and are able to pay any reasonable fee.

In most communities it is generally desirable that the patient be informed in the first instance of the probable cost of treatment. Especially is this true where patients are unfamiliar with the nature of treatment and what it involves. As it is not always easy to estimate the amount of time required for the treatment of a given case, it is generally wise to quote a minimum and maximum fee. If, for example, one regards his time as worth ten dollars per hour, and it is believed, after a careful examination including a radiographic examination, that the case will require ten or twelve hours in treatment, a fee of one hundred dollars to one hundred and fifty dollars may be quoted. When such fees are quoted, note of same should be made on patient's history chart. It may also be desirable, in some instances, to write the patient confirming the oral statement, meanwhile keeping for one's files a carbon copy of the letter. As there are relatively few cases requiring ten or twelve hours for treatment, it will be obvious that in no case is the cost excessive, no matter what one's fee may be, as based on an hourly charge.

THE COST OF NEGLECT

It will also be obvious that the cost of neglect will in every case exceed the cost of treatment, for failure to accept treatment will in time result in the loss of the teeth. There are few, if any, cases in which the direct cost of extraction and replacement will not be greater than that of treatment. And who can estimate the indirect cost as represented by teeth, the supporting tissues of which are involved in a chronic infection, and which as inevitably as the tides ebb and flow, unfavorably influence to some extent the health and well-being of the individual. From an economic standpoint, no patient can afford to neglect treatment, which, if given early, is never expensive and in no case need be prohibitive. Efficiency in the use of one's time and skill in instrumentation will go a long way toward solving the economic problems of periodontia.

While engaged in writing the foregoing, the author made a call on one of his medical friends living in a small New England town, who exhibited a full set of artificial teeth which he was wearing and for which he had paid one of the two dentists in town one hundred dollars. That which interested the author in this incident was not the fact that the fee of one hundred dollars represented a cost in excess of that involved in the treatment of many cases of periodontoclasia, but that a physician who knows the importance to health of good dentistry and who is able and willing to pay for it, should find himself practically toothless when only a little past middle life. There can be only one explanation, and that is that reliance had been placed upon repairative dentistry, instead of being placed where it should have been, on prevention. The result was disaster.

Unfortunately this case represents the condition of many patients who are more or less regular patrons of the dentist. Much of this is due to failure in caring for the supporting

tissues of the teeth. One by one the teeth have become involved in periodontal disease, and one by one they have been lost. The end was artificial teeth.

This should not be, and the fact that it is so is a serious reflection upon dentistry. It means nothing less than that the patient, in spite of his own care, in spite of the time and money spent on repairative dentistry, is in the end penalized by the loss of his teeth. And if the whole cost of that kind of treatment could be known, including the cost of ill health from diseased teeth, it would be found that repairative dentistry costs dollars where preventive dentistry costs dimes. And it would also be found that, as a matter of economics, it is not periodontic treatment that is expensive but the lack of it.

THE ECONOMIC VALUE OF PREVENTION

Mr. George A. Miller, writing for the New York *Evening Post* and quoted in the *Literary Digest* for December 17, 1927, makes the statement that "the dentists of the United States could, if properly financed and properly trained, do more to insure the health, wealth and happiness of our 115,000,000 people than any other profession; they are the guardians of the health of the people, but neither the people nor many of the dentists realize their importance to the economic welfare of the country." He says further: "Preventive dentistry is in its infancy; in fact, it is hardly born yet. The powers that be in the profession are just beginning to think about the problems. Last year, the income of the people of the United States amounted to the enormous total of $90,000,000,000, and of this amount $37,530,000,000 was spent for food. This figure is estimated as 41.7 per cent of the entire cost of living. If the American people ate $37,530,000,000 of food last year, then just think of what it would mean to them if their teeth, which pre-

pared that food for the action of the chemical reagents in the human body, were in prime condition."

It will be noted from the foregoing quotation that the food bill of the people of this nation is colossal, representing almost one-half of the entire income of the nation. If as a result of diseased and defective teeth, one-tenth of the food consumed is unfit for digestion and assimilation (which is a conservative estimate), the direct cost to the people of the United States amounts to the enormous figure of more than three and three-quarters of a billion dollars, more than enough to pay the entire expenses of the Federal Government per annum. And who can estimate the indirect cost as represented by illness, loss of production, premature death, etc.

In a report submitted to the International Conference on Social Work in Paris in 1928, Dr. Homer Folks of New York made the statement that the cost of sickness to the people of the United States exceeded fifteen billion dollars annually. He estimates the total cost of illness up to the day of recovery or death at $3,729,925,396, or $31.08 per capita per annum, and the loss in future net earnings of persons dying prematurely at twelve billion dollars, or an annual loss of $131 per capita. He states further, that while this amount is being spent on illness, seventy-six million dollars only are being expended annually on the prevention of disease. In the city of New York, the direct cost of caring for the sick amounts to $150,000,000 per annum, of which $8,500,000 is expended by public-health agencies. The indirect cost, as represented by loss of income, amounts to an additional $75,000,000. Its ratio of expenditures for the cure and prevention of disease is 18 to 1. It must therefore be evident to anyone that the economic problem involved in the conservation of health is not the cost of medical and dental treatment, enormous

though these may be, but the lack of such treatment; that, in reality, the problem is largely one of education: teaching the public to understand that the cost of neglect is vastly greater than is the cost of treatment; and educating the medical and dental professions and their patients to realize that the cost of illness far exceeds the cost of prevention.

CHAPTER XXIII

CLINICAL CASES

There is perhaps no better way in which to indicate what results one may expect in the treatment of disease than by a report of cases that have actually been the subject of treatment. In this way, one sees more clearly the possibilities and limitations of treatment and obtains a knowledge of the subject that no amount of theoretical teaching can give. The clinician naturally wants to know what can, and what cannot, be expected in the way of results if he follow certain instructions. This can be indicated in no better way than by the recital of cases that have been under treatment and subsequent observation for a period of time. It is with this in mind that the following cases are reported. They all represent more or less extreme and unusual cases, it being believed that if it can be shown that good results can be achieved in such cases, it will be self-evident that those less seriously involved will by so much be more amenable to treatment.

A CASE ELEVEN YEARS AFTER TREATMENT

This was an advanced case of periodontoclasia in the mouth of a physician thirty-three years of age involving all the teeth, and characterized by marked suppuration and loosening of the teeth. No radiograms were made of the case at the time of treatment, as at that period in his experience the author did not make a radiographic examination a universal rule as he does at present. Treatment was begun on January 19, 1918, and continued to March

26th of the same year. Altogether seven hours were spent in instrumentation and prophylaxis. No grinding was done on any of the teeth. The patient left for the war within one week after treatment and was not seen again until September 30, 1921. No treatment had been given him in the interim, but the lesson in toothbrushing and gum stimulation had been thoroughly learned and conscientiously practiced. Except for the upper right second molar which was subsequently extracted, all teeth were firm, all pockets obliterated by reattachment, and the gums were entirely healthy. With two or three prophylactic treatments per year since, the periodontal tissues have remained in perfect health to date. There is considerable gum recession about some of the teeth, but at no time since treatment has there been any sensitiveness of the exposed root surfaces and no recurrence of the periodontal infection. The patient is a moderate cigarette smoker.

REATTACHMENT UNDER ADVERSE CONDITIONS

This was the case of a woman about forty years of age who had already lost several teeth which had been replaced by fixed bridges. Several of the remaining teeth were involved, though not all of them. The maxillary incisors, especially the centrals, represented an advanced stage with extensive resorption, but with relatively little gum recession. Treatment was begun February 1, 1921, and continued to March 3d. Altogether six hours were spent in instrumentation. No incision was made in the gums of any of the teeth and no anesthetic was used. Very slight grinding of the lingual surfaces of upper incisors was practiced. At present all teeth are firm and pockets obliterated. There is at present complete reattachment about these seriously involved incisors.

In July, 1922, the case was made the subject of a report

in the Dental Items of Interest by Dr. Ottolengui, a part of which report is as follows: "The central incisors were so loose that apparently they could have been extracted with the fingers. In cases of this nature it is an odd fact that, whereas the natural teeth only slightly disfigure the patient, their replacement by artificial substitutes is most unacceptable because of the great space to be occupied by two teeth. Fig. 11 was taken of the right central incisor because it seemed the more diseased of the two. (Fig. 57.) Desperate as the radiographs showed the case to be, I referred the patient to Dr. Merritt who undertook treatment because of the urgent appeal of the patient. Figs. 9, 10 and 11 were taken on January 19, 1921. Figs. 12 and 13 were taken April 22, 1922, and are presented for comparison. At the present time these teeth are perfectly firm in their sockets in spite of the very slight attachment of alveolar bone. It is of extreme interest, I think, to note the well-defined, ragged edges of the alveolus prior to treatment and to compare the same with the smoothness of the same area at the present time. But I would particularly call attention to the denser bone which now encapsulates the root ends. (Fig. 58.) Certainly we cannot call this 'sclerotic bone,' though perhaps some would. If so, what harm does this 'sclerosis' effect? Would it not be more in consonance with what we know of repair in general to call this 'scar bone' a condition to be desired rather than feared?" Attention is especially called to this feature of the repairative process; namely, the marked increase in the density of the remaining alveolar bone. This is an impor-

FIG. 57.—Radiograms of incisors, taken by Dr. Ottolengui, representing condition when treatment was begun and referred to by him as Figs. 10 and 11.

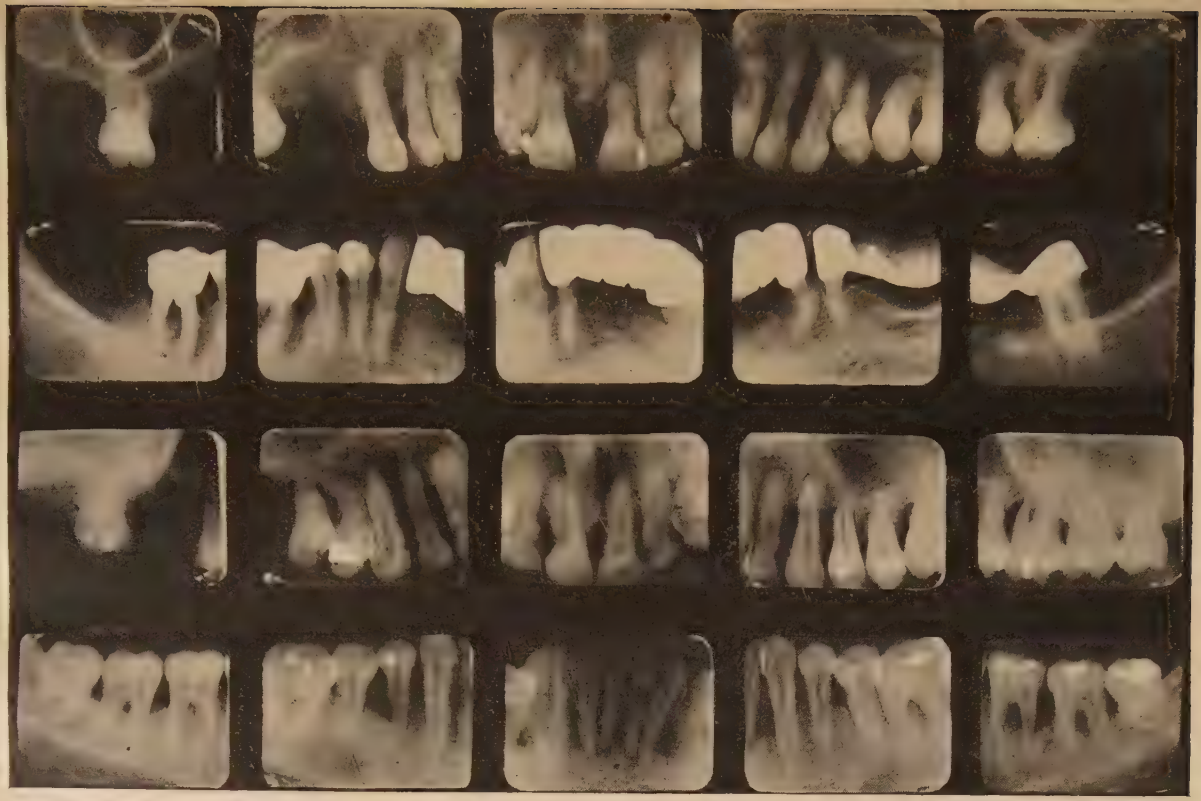


FIG. 58 (upper two rows).—Radiograms taken February 15, 1929, more than 8 years after treatment. Note the density of bone about the incisors referred to by Dr. Ottolengui.

FIG. 59 (lower two rows).—Radiograms of patient with diabetes. All teeth vital and root surfaces extremely sensitive.

tant factor in the stabilizing of all teeth that have been successfully treated for periodontoclasia. It is this increased density, rather than the re-formation of new bone, that explains in part the firmness with which loose teeth are held following treatment, though in some cases both may occur. The reattachment of the gums is also an important element in supporting loose teeth and, in the above case, probably the most important.

Attention is also called to Dr. Ottolengui's observation regarding the changes which took place along the alveolar border following treatment. What had been a ragged and uneven outline of alveoli became smooth, dense and relatively symmetrical. This occurred without any curettement whatever of the alveolar border during treatment, indicating that such treatment is, in most instances, unnecessary. This has been borne out by repeated experiences.

This case was exhibited at a clinic given in 1927, at which Dr. F. V. Simonton and several others were present, as one showing complete reattachment under adverse conditions.

PERIODONTOCLASIA COMPLICATED BY DIABETES

This was an interesting case, because the patient, a man of about thirty-five, had diabetes. As will be evident from the radiograms, periodontoclasia in an advanced stage was present. (Fig. 59.) An unusual feature of the case was the *extreme sensitiveness* of all root surfaces. They could not be touched by an instrument without acute pain. It was the most exaggerated case of this type ever seen by the author. Subsequent examination, as reported by the patient, showed a blood sugar of over 400 mmg. per 100 c.c. Urinary analysis revealed the presence of between 6 and 7 per cent of sugar. Except for the highly sensitive root

surfaces there was, on clinical examination, nothing unusual about the case.

The exaggerated nature of the sensitiveness was probably due to the excessive amount of blood sugar, for in diabetic cases, as pointed out by Winnett, glucose is carried by the blood stream to every tissue in the body, which in cases of periodontoclasia not only creates a favorable culture medium, but directly irritates the exposed cemental surfaces by its presence in the blood stream. If hypersensitiveness of cemental surfaces should be found to be a diagnostic symptom in cases of diabetes, it would be a matter of no little importance as an aid to the dentist in his diagnosis. It is a matter at least that should be kept in mind, and in cases of extreme sensitiveness, an examination for urinary and blood sugar should be made. The author's experience is too limited to say more. He has found it to be true in three cases since his attention was first called to this type of case less than two years ago. In a search of the literature on the subject it was found that Block had reported having observed the same symptom.[1] It is hoped that others in their observations of diabetic cases of periodontoclasia will report their findings on this point.

Because of the extreme sensitiveness of the involved root surfaces, subgingival curettage was impossible without an anesthetic. Accordingly conductive anesthesia was induced by the use of novocaine which, according to Ersner,[3] is the method of choice. Complete desensitization was accomplished in this way. Four treatments aggregating four and one-half hours were given in February, 1928. The response was immediate though less satisfactory than usual. There was complete cessation of pus, the gums took on the appearance of health, and the loose teeth became noticeably firmer. The sensitiveness improved but did not entirely dis-

appear. As the patient lives at some distance from New York, he was not seen for a reëxamination until June 14, 1928. At that time reattachment had not taken place though the improvement was marked. The patient was advised that further treatment would probably be necessary. This case showed none of the grayish-white areas on the gums with their underlying highly inflamed and raw surfaces sometimes seen in diabetic patients.[1] The author has seen several such cases, but has not observed that the teeth were excessively sensitive. In a letter dated August 26, 1929, the patient states that his diabetic condition is much improved. Regarding his periodontal involvment he says: "My teeth and gums have not given me any further trouble. The gums have not grown over the necks of the teeth, though that condition is much improved also. They are pink and healthy and are not affected by heat or cold, etc. The sensitiveness has gone for the most part, though there are one or two spots still. All in all it is quite a comfortable mouth to live with."

PERIODONTOCLASIA ASSOCIATED WITH SYPHILIS

This patient, a young man about thirty, had been under treatment for a couple of years for syphilis, but recent Wassermann tests had been negative. Examination of mouth revealed a typical periodontoclasia with an abundant pus discharge. (Fig. 60.) Two chronic periodontal abscesses with separate fistulous openings were present over the maxillary central incisors. The case was treated between December 9, 1927, and January 17, 1928, by subgingival curettage and without the use of an anesthetic. No incision was made in the gums. Following curettement the discharge of pus ceased, as did most of the objective symptoms. The periodontal abscesses cleared up after the first treatment of the involved teeth and have never reappeared.

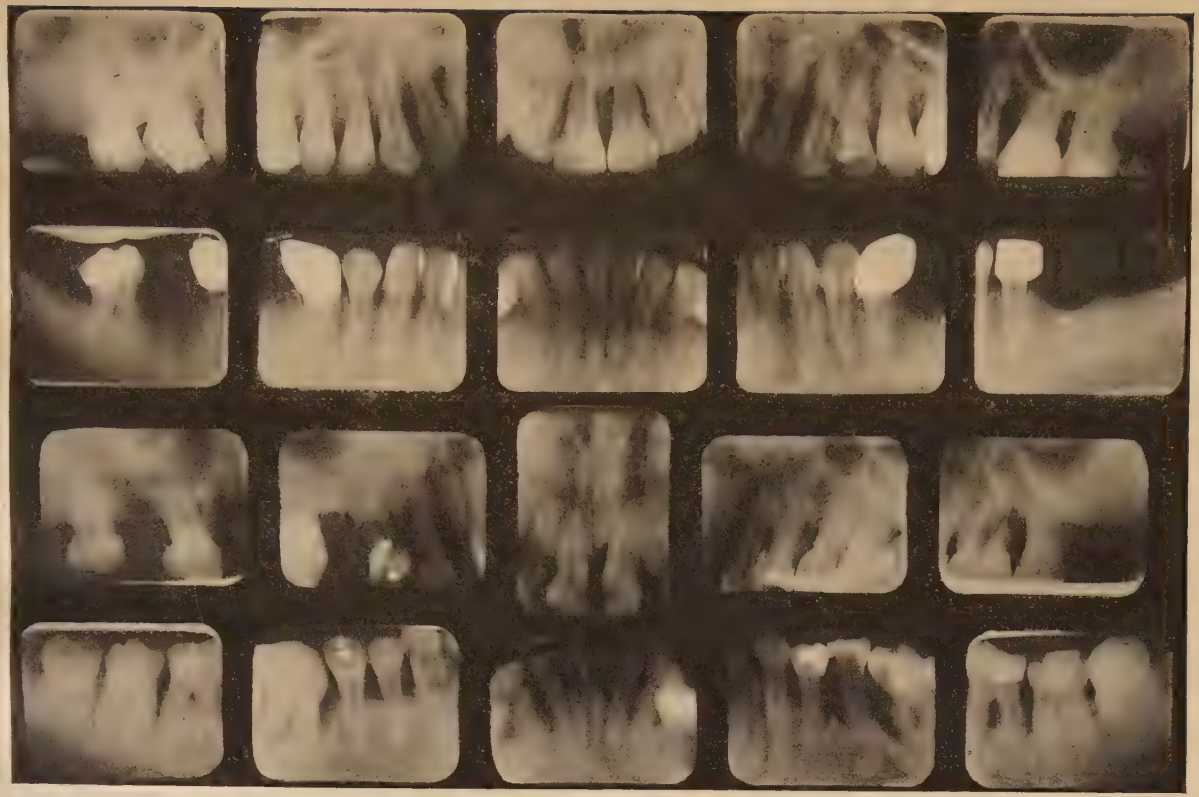


FIG. 60 (upper two rows).—Note loss of bone about central incisors, each of which presented with a chronic periodontal abscess with separate fistulus openings.

FIG. 61 (lower two rows).—Note the extent and irregularity of bone resorption.

Slight grinding of cusps was practiced on a few of the weakened teeth to relieve them of occlusal stress during convalescence. The patient was last seen September 20, 1928, at which time he had maintained the improvement made, though complete reattachment had not taken place and his personal care was not as good as it should have been. He also smokes to excess.

PERIODONTOCLASIA AND HAY-FEVER

This was one of those obstinate cases of periodontoclasia that tax to the utmost the resources of the operator. (Fig. 61.) The patient was a woman about thirty-eight years of age who appeared to be in good health except for a chronic and persistent hay-fever. Treatment in the form of subgingival curettage was begun on November 5, 1928, and with an interruption of nearly two months, was continued to May 9, 1929. Because of its obstinate character thirteen and one-half hours were spent in treatment. The gum was resected about several of the upper posterior teeth, and the occlusion relieved from excessive stress by grinding. During treatment, the hay-fever symptoms disappeared completely for a period of several weeks, when they again reappeared though in less violent form. It is too early to give a complete report on this case. When seen on June 14, 1929, the improvement was all that could be expected: the gums had taken on a normal color, there was no further discharge of pus which had been a marked symptom in this case, and the teeth were less sensitive and obviously firmer. It is reported because of its obstinate nature and because of the associated hay-fever symptoms. There was nothing in the patient's history or physical examination to explain the case. Except for her very obstinate hay-fever which has resisted repeated efforts at treatment, she appeared to be in excellent health.

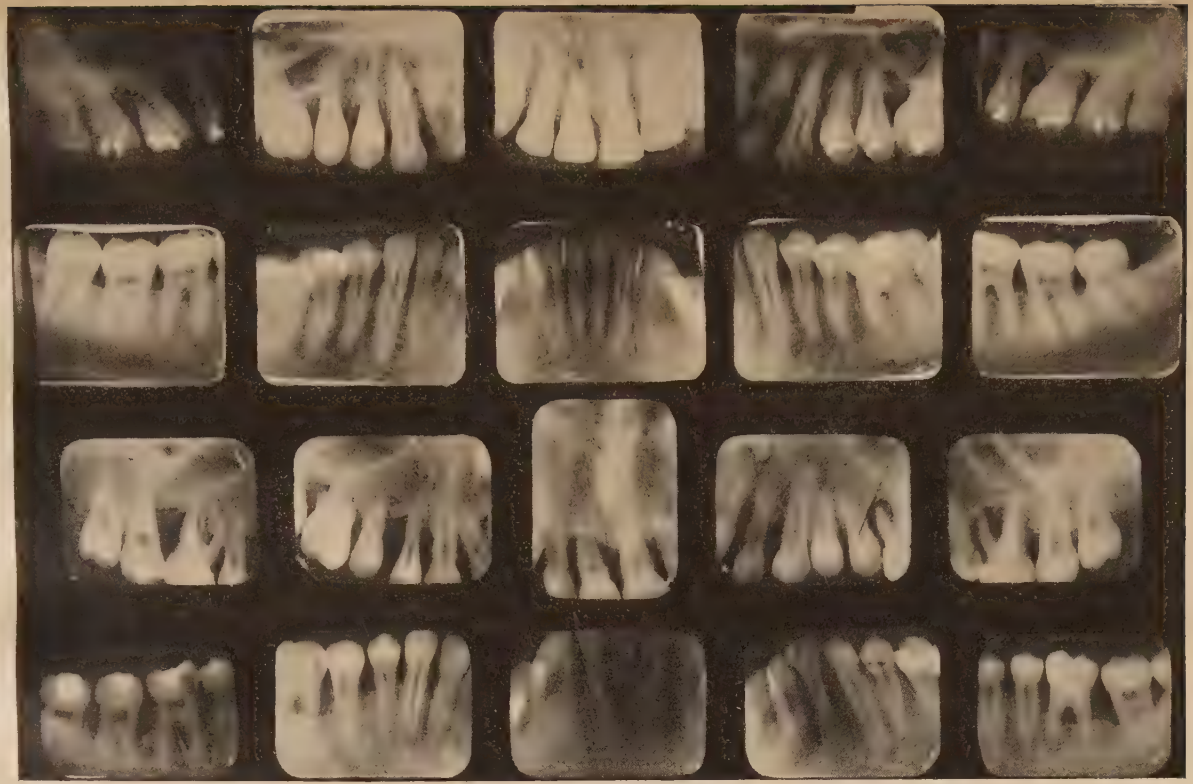


FIG. 62 (upper two rows).—Observe amount of bone loss about upper first molars.
FIG. 63 (lower two rows).—Radiograms of patient 25 years of age. Note extent of bone loss; also supernumerary tooth between upper centrals.

A TYPICAL CASE OF PERIODONTOCLASIA

This patient, a woman thirty-five or forty years of age, had all her teeth, which were in normal occlusion and unusually free from caries. (Fig. 62.) Her personal care had been fair and she had always been a regular patron of the dentist. There was nothing in her general condition to explain the periodontal disease. As represented by the radiograms it will be observed that the condition of alveolar absorption is one that is typical in these cases; some teeth seriously involved while others show relatively little bone loss. In one other respect it was also typical of many cases; it was difficult to explain its development. The patient was in good health, her diet was reasonable, and the hygiene of her mouth was above the average. Her occlusion was normal and well balanced. The only local conditions which might offer an explanation were periodontal tissues that were functionally inactive and dental care which had not sufficiently taken into consideration the condition of her periodontal tissues. And right here let it be said, without criticism, that this latter factor is probably the most common explanation of many cases. This patient had gone to her dentist at regular intervals all her adult life, who, finding no caries, had dismissed her without giving much attention to the health of the supporting tissues of her teeth.

This case was treated by subgingival curettage between April 11 and June 4, 1927. Altogether six and one-half hours were spent in treatment. In addition to the usual treatment, the gum was excised on the buccal surface of the right mandibular first molar as the alveolar process had been partially destroyed in the bifurcation, a condition found to be worse than was indicated by the radiogram. The gum was likewise excised, this time circumferentially, about the right maxillary first molar. It will be observed,

by examining the radiograms, that the left maxillary first molar was about as badly involved as was the one on the right side. It was decided to excise the gum tissue about the one molar and to treat the other by the more conservative method of subgingival curettage with a view to comparative observation. Several of the teeth were so weakened that they had to be ground to relieve occlusal stress, their condition being such that they were incapable of withstanding their normal load.

The response to treatment was most satisfactory from the beginning. All pus discharge ceased as each tooth was treated, the gums resumed their normal color under the combined action of operative and postoperative treatment, and the whole mouth took on the appearance of health. At this time, more than two and one-half years after treatment, this condition continues with the added firmness of the teeth and partial reattachment of the gums. Those about the upper left molar are hard, pink and healthy. They have partially shrunken back as health has been reëstablished about this tooth, though reattachment is not complete and probably never will be. The important thing about this, as about all such cases is, that health in the periodontal tissues has been reëstablished and the patient is entirely comfortable and conscious likewise of the improvement. The weakened teeth, the receded gums, the deepened crevicular spaces, etc., remain as the scars of disease. They all bear testimony to the transcendent value of prevention, or, failing in this, the desirability of early treatment.

UNUSUAL SYMPTOMS FROM OCCLUSAL STRESS

This is a case representing unusual symptoms from occlusal stress. The patient, a man forty-one years of age, presented himself, complaining of neuralgic pains in the left

infra-orbital region. It had existed intermittently for about two years. At times it was so severe as to be disabling. He had consulted several physicians believing it to be "neuralgia." In his effort to obtain relief he had traveled several hundred miles to consult a nerve specialist. There had never been any pains in the mouth. An examination showed all teeth to be present. They had always been relatively free from caries. Except for a superficial gingivitis there was no periodontal disease. Teeth were in malocclusion, class one. The mandibular left first bicuspid was in marked malocclusion with its antagonist in the maxilla, both of which were almost entirely outside the normal arch. Except for the fact that they were malrelated to each other, there was nothing to indicate that these teeth were in any way responsible for his symptoms. Having in mind a similar case though with less definite symptoms, the two bicuspids were entirely disoccluded. As there was at the time nothing to indicate whether this would afford him permanent relief, he was asked to report in a month. At that time he reported having had complete relief, and in the nearly two years which have since elapsed there has never been a recurrence. The interesting feature of this case was the definitely located symptoms in the malar bone, clearly defined and at times very severe, without there ever having been any symptoms whatever in the mouth. The author has seen similar cases but never any in which the pain was so definitely located or so acute. The usual symptom in these cases is a feeling of discomfort coincident with, or following the use of, the teeth, not definitely located, but radiating out into the areas supplied by the second or third divisions of the trigeminus. The patient usually relates the symptoms to the teeth. Usually very little grinding affords relief.

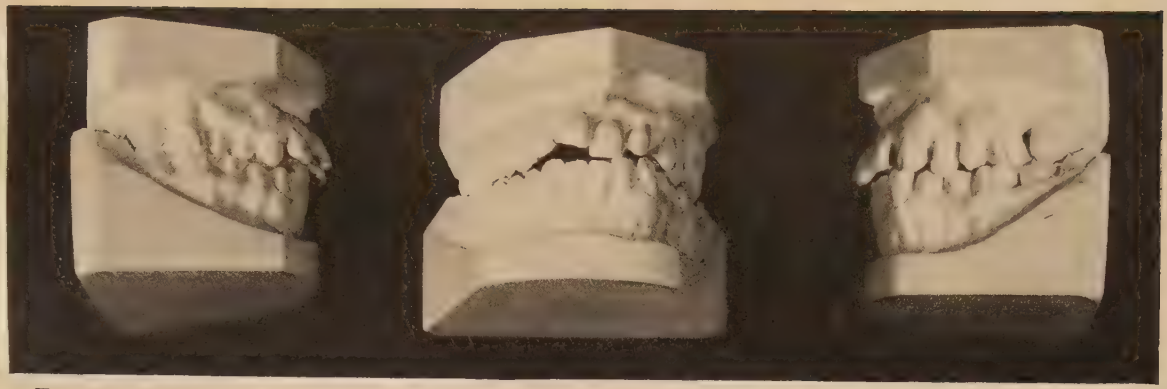


FIG. 64.—Models showing malposition of teeth due to looseness from bone loss. See radiograms, Fig. 63.

PERIODONTOCLASIA IN A PATIENT OF TWENTY-FIVE

This was an unusual and interesting case of periodontoclasia in an advanced stage, in a girl twenty-five years of age. (Fig. 63.) She was five feet, four inches tall and weighed 118 pounds. She claimed to be in excellent health, but was referred to her family physician for a complete physical examination. Inquiry regarding her diet revealed it to be badly balanced and subnormal in quantity. In her desire to maintain a "boyish figure" she was clearly undernourished. She also smoked to excess. Examination of the teeth showed all to be present, but in malocclusion due in large measure to their having changed their position as a result of the periodontoclasia. (Fig. 64.) Several of the teeth were markedly loose and the four first molars were all pulpless. She had been taken by her physician to an oral surgeon of excellent reputation who had advised complete extraction. The report from her physician being negative, treatment was begun February 9th and continued to May 16, 1929, during which time twelve and one-half hours were spent in subgingival curettage. No anesthetic was used and no gum excision practiced. She was at the same time put on a diet consisting of one quart of milk per day, raw fruits and leafy vegetables, plus her usual diet. At the completion of treatment on May 16th she had gained twelve pounds in weight, the discharge of pus had ceased and the gums had taken on the appearance of health. She was then seen in consultation with Dr. A. LeRoy Johnson with a view to orthodontic treatment. Because of the unusual condition, and being dissatisfied with the report from her family physician, he advised that another physical examination be made by a second physician which was done. This was also negative, though he expressed the belief that her restricted diet might have been a factor

in etiology and approved of the changes that had been made.

These two negative physical examinations made by competent physicians working independently, indicate the elusive nature of some of the possible etiologic factors met with in cases of periodontoclasia. There was every reason to believe that in this case there were etiologic factors other than those appearing in the mouth, but notwithstanding the fact that one of the physicians making the physical examination had been chosen because of his known competency for such an examination nothing was found to indicate anything abnormal. It may be said in passing that this is the usual experience in these cases. This may mean factors exceedingly elusive or it may mean that the systemic factor is lacking altogether in most cases.

Under advice from Dr. Johnson the pulpless molars are to be retained during orthodontic treatment, at the completion of which they and the upper left second bicuspid are to be extracted. The patient is now under his care though she is seen at regular intervals for prophylactic treatment. At present, excellent progress is being made, though of course it is too early to give a final report. It has been included in these case reports because of its unusual interest and because there was nothing in her condition as established by two physical examinations made independently, that offered any clew as to why so advanced a case of periodontoclasia should occur in a person twenty-five years of age. The only factor outside of the mouth offering any explanation was diet deficiency. The local conditions were not unusual. It is one of those cases of which, like Tennyson's "Flower in the crannied wall," it might be said, "If I could understand what you are, root and all, and all in all, I should know what God and Man is."

REFERENCES

[1] Bloch, H., 1922, "A Classification of Oral Manifestations of Systemic Diseases According to Type," *Journal American Dental Association,* vol. 9, p. 151.

[2] Editorial, 1910, "Gingivitis in Diabetes," *Journal American Medical Association,* vol. 52, p. 1164.

[3] Ersner, W. E., 1926, "Relation of Diabetes to Dental and Oral Surgery," *Dental Cosmos,* vol. 68, p. 279.

[4] Williams, J. B., 1928, "Diabetic Periodontoclasia," *Journal American Dental Association,* vol. 15, p. 523.

[5] Ziez, J., 1915, "Statistical Observations on Diabetes and Pyorrhea," *Dental Cosmos,* vol. 57, p. 102.

CHAPTER XXIV

CONCLUSIONS

As one reviews the history of periodontia as it is represented in the etiology, pathology and treatment of periodontal diseases, certain facts stand out more or less clearly. First and foremost among these is the need of research. Anyone who has given the subject any thought whatever cannot have failed to be impressed with "how far the unknown transcends the what we know." Not only does this obligation for further research rest upon the individual working in the laboratory with microscope and scalpel, but the clinician also must share in the responsibility. The research worker in the laboratory has in his field far outstripped the clinician at the chair. He has been engaged in a work for which the latter is not fitted by temperament, training, or experience. And he has done and is doing his part well. Too much credit cannot be given to such men as Box, Gottleib, Howe, Noyes, Orban and Simonton, only to mention a few who are outstanding in the field of periodontology. Without their work, the clinician could not have hoped to make the progress which he has made. They are the advance guard, so to speak, who blaze the trail for the man who stands on the firing line of daily practice. But as already pointed out, the latter has his part also in the research program. This consists in more carefully recording his clinical observations and experiences than has been his custom in the past, and making them available to others working in the same or related fields. Much

information that would be exceedingly worthwhile could be obtained in this way. Models of cases should be made by the clinician more often than is usually done. Their scientific worth, when made in considerable numbers, would be invaluable. To obtain best results, these should be made from plaster impressions, which, of course, is not always easily done. Nevertheless their value would more than justify the effort made. In these and other ways, the observing clinician can do much more than he is now doing to extend the boundaries of knowledge in the field of periodontology, an obligation which he is urged to accept.

The second fact to impress itself upon the mind as one studies clinical periodontia and reflects upon how large a part neglect plays in the promotion of periodontal disease, is the great need of a more general application to daily practice of the knowledge already available in the field of periodontology. As has been pointed out, it is entirely possible to prevent or cure, by early treatment, practically every case of periodontoclasia if the dentist, in the care of his patients, would avail himself of the knowledge already at hand. If one will study the case reports in the foregoing chapter he will observe that they all represent advanced cases in relatively young patients. The history of every case reveals the fact that the patient had been a regular patron of the dentist, yet each presented himself with a perfectly typical case of periodontoclasia so far advanced as to give an uncertain prognosis. In every instance, several years had elapsed since the beginning of the disease and at no time during those years would there have been any difficulty in diagnosis had the knowledge, easily available, been made use of. The failure to do this constitutes the most serious present-day problem in periodontia, and one which should have the attention of every member of the dental profession. This is not said in any spirit of criticism. It is not altogether

the individual dentist who is at fault in this. In many instances, he received no instruction whatever in periodontia in his undergraduate days, and very little in the way of postgraduate work has ever been available. These facts, with the conflicting opinions on the subject which so often find their way into current literature, have resulted in bringing about a state of mind alternating between confusion and indifference. What is needed to correct this unfortunate situation is the inclusion in every dental curriculum of a comprehensive course in periodontia. This, the author is glad to report, is being done. And in addition to this, an effort should be made to help the practitioner who, like the author, was denied the privileges of a present-day dental education. There are several ways in which this can be done. One is the inclusion in the programs of state and national organizations of courses of instruction in practical periodontia. The importance of this has been recognized by the American Dental Association in recently creating a section in periodontia in that organization. Many state societies are also to be commended for the place which they are giving on their programs to periodontia.

Another way in which to meet this need is by intensive postgraduate instruction, preferably under the auspices and supervision of the university dental schools. Some efforts in this direction have already been made and others are needed. These should place the emphasis on prevention, at the same time including in their instructions a comprehensive course in the principles and practice of periodontia. A few weeks of intensive study, including clinical instruction in operative technique, would be of inestimable help to the postgraduate student. Another way in which the busy practitioner may receive assistance in the field of periodontia is by a study of its literature. Every phase of the subject is being represented by excellently written

articles in the current dental magazines, many of which are worthy of careful study. Textbooks covering the entire field are also to be had by those wishing to avail themselves of the information they impart. With their aid and such assistance as is afforded by clinical demonstrations, the busy practitioner can do much to make amends for his lack of undergraduate instruction. The great need which exists in this phase of periodontic practice makes it a subject to be seriously considered by every practicing dentist. It is not further research, important though that is, but the application to daily practice of the knowledge already available, that represents the great outstanding need of present-day periodontia.

Equally important in the prevention, control and treatment of periodontal diseases is the need of more comprehensive dental examinations. No examination can be regarded as complete that does not include a radiographic survey of both jaws; a thorough clinical examination of the teeth and periodontal tissues for caries, defective contact points, malocclusion, occlusal stress and gingival abnormalities, plus inquiry as to the patient's general health, diet, etc. The radiographic examination should be made at intervals of a year or two, if the best results are to be obtained. It is realized that economic conditions may make this impossible of universal application, but it should be made the rule in every dental practice and applied whenever conditions permit. Treatment based on an incomplete examination can never be entirely satisfactory. The fact that it is so often done accounts for many failures in daily practice and explains also one of the great obstacles to prevention.

And finally, as one surveys the field of periodontology and reflects upon the nature of the diseases which involve

the periodontal tissues, he cannot fail to be impressed with the importance and desirability of prevention and early treatment. Because of the tendency of these diseases to break down and destroy the supporting tissues of the teeth, their progress is attended by conditions that to a certain extent are irreparable. The resorbed alveolar process, the receded gingivæ, the exposed and unsightly root surfaces, the weakened and loosened teeth, can never be restored to their original conditions no matter what the treatment may be. Cases that are far advanced may be cured in the sense of restoring them to health, but that is all; the destroyed tissues, except in rare instances, will never be replaced. For these reasons, no treatment can ever be entirely satisfactory.

Another factor in favor of prevention is the ease with which it can be accomplished. With the coöperation of the patient, and the intelligent application to daily practice of present-day knowledge regarding periodontal disease, prevention could be made the rule. And this very desirable object can be achieved by measures so simple that anyone interested in doing so can apply them. These consist in establishing functional activity, supplemented, if need be, by artificial stimulation and the continued maintenance of a high standard of mouth hygiene. By their joint action, if persisted in throughout life, most periodontal diseases could be prevented altogether. And in the whole field of dental practice, there is no service more worth while.

As one pauses at the conclusion of his survey of periodontology, and reflects upon what is required to make it more successful in the field of oral health service, the following facts will be more or less obvious:

First: The necessity of further research in the laboratory and at the chair; Second: A more general application to

daily practice by every dental practitioner, of the knowledge already at hand in the field of periodontia; Third: The need of more comprehensive and more thorough dental examinations; Fourth: The importance of prevention and the relative ease with which it can be accomplished. These four—and the greatest of these is prevention.

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